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THE OPEN SAFETY PIN*

A Consideration of Its Peroral Removal from the Upper Air and Food Passages

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AS APPLIED to a peroral foreign body, there are few greater misnomers than the term "safety pin." So far as its presence in the upper air or food passages is concerned, this type of pin is at once one of the most dangerous and frequent objects to gain accidental access to these regions. The danger arises primarily from the fact that in most cases the pin tends to enter the pharynx or larynx with the point unguarded and held at a wide angle by the spring, thus presenting a hazardous situation in which the delicate walls of the upper air and food passages are subject to pressure and possible perforation by the point of the pin. The frequent occurrence of such accidents is due to the almost universal use of the safety pin in dressing infants — an age group in which this type of foreign body is most frequently seen. In spite of constant medical admonition, mothers and attendants continue to leave safety pins open and with unprotected points within easy reach of babies whose natural instinct is to put them in the mouth. Added to this is the bad example so often set by mothers themselves in holding such pins in the mouth, with the inevitable result that youngsters tend to imitate this indiscretion. Occasional accidental swallowing of the pin by an adult may result from this pernicious habit.

I believe that this situation warrants closer attention from the pediatrician and the general practitioner, who will so frequently be the first to see such patients. For this reason I am presenting some conclusions drawn from personal experience with regard to clinical observations and secondarily to the operative technic involved in the peroral removal of these foreign bodies.

In a recent article, Clerf[1] reports that of over 825 foreign bodies removed endoscopically, 77 were safety pins. In 59 cases, or 77 per cent, the pin was removed from the food passages; in the remaining 18 it had lodged in the air passages. In a personal series of over 150 peroral foreign bodies, 25 have been some form of safety or clasp pin, 90 per cent of which were in the food passages.

Because of this experience with the removal of such pins from the upper air and food passages, I have come to believe that no specific operative technic is applicable in all cases. Chief consideration must be given to the location of the pin and the relation of the point to this location. As an aid in the visualization of the commonest sites of pin lodgment, there is reproduced in Figure 1 a group of photodiagrammatic x-ray films to show representative situations within which, with slight variations, will fall almost all open safety and clasp pins that accidentally find their way into the air and food passages. Divergencies from these typical positions will of course occur, but slight differences of a centimeter above or below the midesophageal region or a minor tilting of the pin to the right or left has no essential bearing on satisfactory methods of removal later to be discussed. These representative positions will first be considered individually.

LOCATION OF PIN

Hypopharynx

It is in the hypopharynx (Fig. 1*A*), particularly in infants up to the age of one year, that one finds over 50 per cent of all open safety pins. The point is usually upward either to the right or to the left and most frequently buried, at least at the termination of the shaft, in the mucosa of the lateral pharyngeal wall. In this position, the spring is resting just at the mouth of the esophagus and occupies the somewhat narrow constriction known

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as the cricopharyngeal ring formed by muscle fibers that, attached to the posterior surface of the cricoid cartilage, are embedded in the lateral and posterior surfaces of the constrictor muscles. This muscular ring exerts a somewhat sphincterlike action and is recognized as a line of demarcation or boundary with respect to any esophageal foreign body, depending on whether such objects

The relation of the plane of the pin to that of the hypopharynx is of almost pathognomonic significance. Any pin in the hypopharynx will appear, in a true anteroposterior roentgenogram, in its full expanse, with but little if any foreshortening, as shown in Figure 1*A*. For this reason, in a true lateral view, the pin will be seen from the side and hence will show no evidence of details

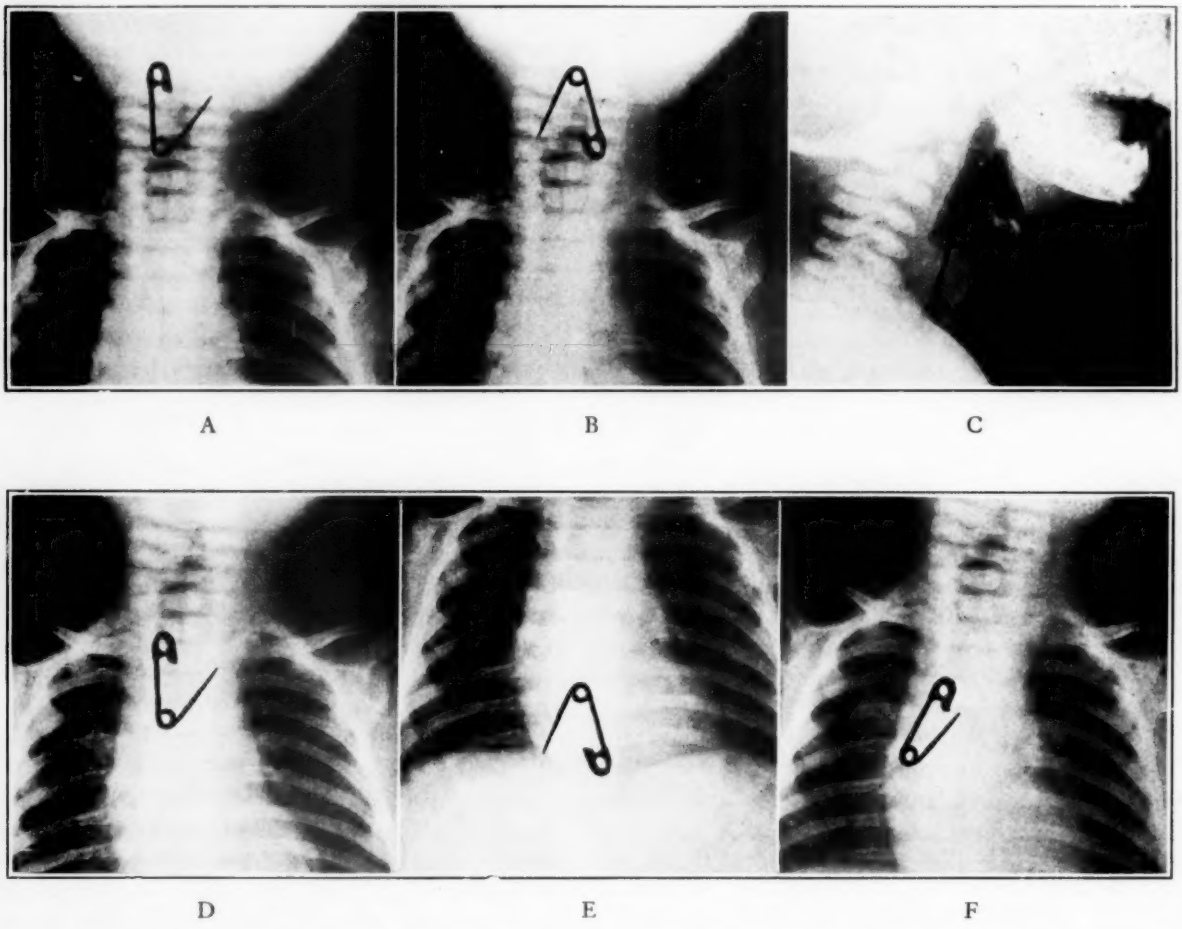

FIGURE 1. *Photodiagrams of Open Safety Pins in Representative Positions in the Upper Air and Food Passages.*

A — *hypopharynx, point up;* B — *hypopharynx, point down;* C — *larynx, point up;* D — *esophagus, point up;* E — *esophagus, point down;* F — *right bronchus, point up. An intentional disproportion between the size of the pin and the roentgenogram has been employed for purposes of illustration.*

are above the cricopharyngeus muscle or below it. This differentiation, as will be seen later, is of the greatest importance in selecting the method of removal best suited to the individual case. The keeper of a pin in the hypopharynx is usually found at a slightly higher level than the point, but slight changes in angulation may leave both keeper and point on a horizontal line. Most frequently, the keeper shaft of the pin will be in a vertical position, the pointed shaft assuming an angulation with the vertical dependent on the degree to which the pin is open.

of expansion or relation of point and keeper to each other. Since an absolutely uniform lateral exposure is difficult to secure, there will, in this view, frequently be a slight divergence between the two shafts, but this appearance is quite distinct from that seen when the pin is lodged in the larynx (Fig. 1*C*).

Lodgment of a pin in the hypopharynx depends somewhat on its size. The larger the pin, the greater the spread at the spring and the less likely it is, particularly in young children, to pass deep into the upper esophagus. In spite, however,

of any relatively high roentgenologic appearance, it is only rarely that the pin can be seen on direct inspection of the throat. When this is possible on forceful depression of the tongue, the top of the keeper may be glimpsed, a point of importance only if x-ray facilities are unavailable or yield negative results.

Far less often a pin in the hypopharynx comes to rest point downward (Fig. 1*B*). This position is at once recognizable roentgenologically, the pin still assuming its typically transverse relation in the anteroposterior view.

Larynx

It is Nature's intention that any appropriate object put in the mouth shall be swallowed, passing into the esophagus and thence into the stomach. To this end, a complicated and beautifully co-ordinated series of muscular movements propels the object along the proper course. For this reason, more than 90 per cent of all open pins that find accidental lodgment in a child's throat are found in some part of the alimentary tract — hypopharynx, esophagus or stomach. During the act of swallowing even so awkward an object as an open safety pin, there takes place a temporary closure of the larynx or gateway to the upper air passages, which prevents any foreign substance from gaining access to this vital and sensitive region. Only occasionally, in an unguarded moment, can a foreign object near the orifice of the larynx, during a deep inspiration provoked by laughing, coughing or crying, evade the swallowing guardian and so be drawn into the upper airway. This situation, which the layman interprets as "swallowing the wrong way," causes such small objects as nut fragments, tacks and other bits of hardware to reach the tracheobronchial tree.

This same explanation accounts for the occasional lodgment of an open safety pin in the larynx (Fig. 1*C*). Since the diameter of the latter is much less than that of the hypopharynx, it is quite understandable why a pin of any except extremely small size meets obstruction to further advance and therefore, fortunately, fails to enter the trachea. Again, as in the hypopharynx, almost all such open pins in the larynx come to rest with the point upward but with the fundamental and diagnostically significant difference that the pin lies in the sagittal plane. Therefore, a view of its details in the plane of expansion is seen only in lateral roentgenograms of the neck. In an anteroposterior film, there appears usually only a single or closely approximated double shadow, and even this is obscured by the density of the cervical spine directly behind it. The assumption of such a position by an open pin in the larynx is owing to the fact that whereas the greatest width of the pharynx and esophagus is in the frontal plane, that of the larynx is in the sagittal plane, and only by conforming to this plane can any appreciable part of the pin pass between the vocal cords. It is at once obvious that this fixed relation of the pin to the plane of the food passage or airway is of the utmost diagnostic value in determining the location of the foreign body. More commonly, an open safety pin enters the larynx with the pointed shaft anteriorly, the point itself protruding either just above the cordal junction in the anterior commissure or just beneath this point and hence below the cordal junction. Depending on the size of the pin, the keeper will occupy a position above or between the arytenoid cartilages, in either event resting against the interarytenoid fold in the midline. Only very rarely will such an open pin enter the larynx point downward, when it will still assume the characteristic anteroposterior plane and will be seen in its expanded position only in the lateral film.

Esophagus

It is in their lodgment in the esophagus proper (cervical, upper thoracic or lower thoracic) that open safety pins create the greatest hazard to the patient and the greatest endoscopic problem for the bronchoscopist. Once the pin has passed the sphincter of the cricopharyngeus muscle, it comes to rest in some portion of the esophagus, most commonly in the region illustrated in Figure 1*D*. Variations somewhat above or below this representative point have no special bearing on the problem of removal and are dependent on such extraesophageal relations as the crossing of the left main bronchus or pressure from the arch of the aorta. So long as such pins do not pass promptly into the stomach, they call for endoscopic removal, and the assumption that they will pass spontaneously is unwarranted by experience. Here again, as in the hypopharynx, the pin will assume its characteristic frontal plane, its detailed relation being clearly evident in anteroposterior roentgenograms.

By far the greatest number of such esophageal pins are found point up. When the reverse is true, there is always a strong suspicion that the pin, advancing spring downward, actually entered the stomach, being subsequently regurgitated back into the esophagus and lodging this time with the point downward (Fig. 1*E*). Only in cases of extremely small baby safety pins in older children or adults would it normally be possible for an open safety pin to advance to the midesophagus through the constricting cricopharyngeus muscle while maintaining the point in advance.

Main Bronchus

It requires no knowledge of peroral endoscopy to appreciate the hazards to the patient and technical difficulty of removal of an open safety pin lodged in the main bronchus, the rarest of locations, particularly if point up (Fig. 1*F*). As a rule, only rather small pins can, through sheer impossibility of passing the larynx, reach the lower bronchial tree, barring always the occasional misfortune of dislodgment from the larynx during attempted removal from the latter. The angular spread of any open pin in the bronchus must of necessity be limited, since the bronchial diameter automatically precludes wide angulation.

Stomach

Finally, open safety pins not infrequently are first discovered to be in the stomach, having, through Nature's providence, passed without accident through the esophagus. Whether, once arrived in this temporarily safe harbor, such pins merit endoscopic extraction is a matter of experience, individual judgment and meticulous study of the subsequent clinical course, and constitutes a subject best discussed in connection with the considerations of removal.

Diagnosis

It is self-evident that the proper treatment of any patient harboring an open safety pin in his air or food passages presupposes a diagnosis of its presence. Consideration must first be given to a history of ingestion or aspiration, or at least of an opportunity for this. In most cases, such a history must come from the parents, almost always the mother or an attendant, less frequently from an older child. Infants or small children can obviously afford no help in such a situation. It is interesting to note that a positive and volunteered story of the swallowing of an open safety pin is much oftener available than that of nuts aspirated into the bronchus, for example. In a personally observed series of 40 cases of such aspirated nuts,[2] the proportion of delayed diagnosis and prolonged sojourn of the foreign bodies before removal was far higher than that of open pins. This fortunate circumstance results, in all probability, from the fact that most persons caring for infants and young children have a fairly accurate idea of the number of pins customarily used in dressing them and of their whereabouts during any moment of changing or undressing. If, during an unguarded moment, the infant procures such a pin, puts it in his mouth and swallows it, it is quite likely that the pin will be missed and a prompt search made for it. With failure to find it, suspicion is very apt to arise that the child has swallowed it. If, at the same time, there are any such untoward signs or symptoms as coughing, gagging, choking and vomiting, the possibility is likely to be still further considered and a physician called. Apparently, perhaps as the result of greater publicity, parents today seem much more conscious of pins than of such a foreign body as a peanut, with which, as evidenced by the above-mentioned series of 40 cases, the time interval between aspiration and bronchoscopic removal is much longer.

Another factor operating to effect prompter recognition of a safety-pin accident is the age of the patient. In a high percentage of cases, this is less than one year, hence patients have not the freedom to come and go and so, of their own accord, to put other foreign objects in their mouths. In the group aspirating some form of nut, the average age of the patient was over three years, obviously precluding a parental supervision comparable to that of infants under a year of age. Nevertheless, there have been patients who have swallowed or aspirated open pins in whom there was no history of the accident and in whom symptoms caused by the pin were, for an unfortunate period of delay, attributed to some more humble cause. The following case, seen at the Bronchoscopic Clinic of the Massachusetts Eye and Ear Infirmary for removal of an open safety pin from the larynx, clearly illustrates this point.

R. N., a 7-year-old boy, previously perfectly well, while attending school suddenly choked, coughed and vomited. Sent home by the teacher, he immediately reported to his grandmother that he had swallowed a safety pin. She, being quite hard of hearing, failed entirely to grasp the significance of this story. On the return of the boy's mother, she found him quite hoarse and, presuming him to have a cold, summoned a physician. For the ensuing 2 weeks the boy was treated by three physicians for laryngitis, since he advanced no further information about the pin. The latter was only discovered when, after 3 weeks from the date of accident, a fourth consultant suggested an x-ray, which clearly showed the open pin in the larynx. The pin was removed by the method described below.

It is thus apparent that any physician called in such an accident must at least keep the possibility of foreign body, uncommon as it may be, in mind as a cause for sudden, unexplained deglutitory or respiratory distress. It is at least important, so far as infants and safety pins are concerned, to inquire whether a pin is missing, if such information is not previously volunteered. To this point of suspicion on the part of the attending physician, I have called especial attention in discussing accidental inhalation of nuts.[2] Fortunately, an x-ray film is an infallible diagnostic aid in determining the presence of an open safety pin, but its use must be preceded by the thought that a

foreign body may be present. In spite of this, numerous cases are on record in which one and sometimes more than one open pin have remained unrecognized in the esophagus, the symptoms being attributed to some gastrointestinal disturbance, and no consideration given to the true cause of the trouble.

With respect to pins in the air passages, as exemplified by the case cited above, hoarseness is an almost invariable accompaniment of a safety pin in the larynx, since the presence of the pin prevents accurate apposition of the vocal cords. If, however, the pin chances to reach the bronchus, the clinical course may be essentially asymptomatic, except for a slight cough. In both the larynx and the bronchus, respiratory obstruction is not usually a marked symptom. The actual encroachment on the airways by such a pin even in the larynx is not great, and strange as it may seem, the surrounding inflammatory reaction provoked by the presence of the intruder may be very slight. This is in marked contrast to the reaction to the presence of a nut in the bronchus, in which, besides acting as an obstructing ball valve, it is apt to set up a severe and aggravating inflammatory reaction, tending to occlude the airway completely.

Knowing of or suspecting the presence of an open pin in the throat, parents and physicians should, above all, refrain from any crude or traumatizing attempts to remove it. Such efforts will only succeed either in driving the pin still deeper in the throat or possibly in inflicting severe trauma to vital structures. This warning is given, notwithstanding the following letter received on the occasion of a previous similar and public admonition:

> I was reading your lecture. When my girl was two years old she got a tin whistle in her throat. I was all alone so took her across my lap and shaked and rubbed her. The whistle came across the floor. My doctor said I saved her life. My aunt's brother got a safety pin in his throat, open. She took a buttonhook and saved him. I would do the same thing again to my two grandsons today. You have to use what help is near.
>
> A Grandmother of Three

Once the diagnosis of the presence of an open pin in the hypopharynx, larynx or esophagus has been made, the patient should be referred to some institution adequately equipped for the safe removal of such potentially dangerous foreign bodies.

Removal

It has already been stated that methods for the removal of open pins in the air and food passages must be selected according to the particular problem in hand. Jackson,[3] in a recent textbook, lists sixteen different possible technics for removing safety pins. It is manifestly impossible for an endoscopist working in any but the largest endoscopic clinic to be master of such a multiplicity of procedures, and it has been my experience that in almost all cases reliance on a much smaller number of maneuvers, thoroughly studied and frequently practiced on a manikin or in the surgical laboratory, yields satisfactory results. Unquestionably, very unusual situations require specialized variations in technic, but the description of the following methods will, it is believed, prove adequate as a guide for removing the great majority of open safety pins encountered over a considerable period.

Preliminary to any attempt at removal, it is imperative to make a most careful roentgen-ray study of the patient, clearly establishing the locality of the pin and its relation to anatomic structures. From this study, it should be possible to ascertain, in comparison with a graded series of standard pins, the size of the pin in question. Still more helpful, if available and dependable, is an exact duplicate of the pin brought in by the parent or patient.

Anesthesia

The question of anesthesia has long provoked controversy and disagreement among leading endoscopists. Jackson,[3] backed by a wealth of experience, has persistently been an advocate of the policy of giving no anesthesia to children and only local anesthesia to adults. The merits of the various forms of anesthesia available for all endoscopic procedures are beyond the scope of this paper. It has been my experience, so far as the removal of an open safety pin is concerned,—particularly in the esophagus and in infants and very small children,—that there are more advantages than disadvantages accruing from the use of a general anesthetic. With foreign bodies in the larynx and in any patient in whom there is a question of respiratory embarrassment, I should counsel the avoidance of any form of general anesthesia. Otherwise, with appropriately sized instruments, with a competent anesthetist, and with the exercise of utmost gentleness of manipulation, I believe that the resultant relaxation of the esophageal musculature is an advantage that far outweighs any objection to general anesthesia.

Pins in the Hypopharynx

Point down. In the few cases in which the pin lies point down in the hypopharynx, the mechanical problem should not afford difficulty. Either

a laryngoscope or a small-sized esophageal speculum is used to expose the lower portion of the hypopharynx. The spring of the pin is viewed under direct vision and grasped with alligator forceps, preferably one in which the tips of the jaws carry a small projection. When these projections close over the spring of the pin, they do not fix it but permit it some latitude in lateral rotation, or what may be termed "toggling,"

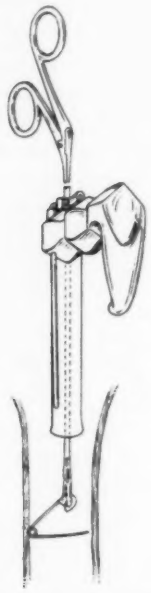

FIGURE 2. *Method of Extraction from the Hypopharynx. The keeper of the pin is grasped; this allows the pointed shaft to follow the line of least resistance.*

at the same time avoiding any tendency for an insecure grasp with possible slipping off of the pin. This toggling permits the pin, during withdrawal, to adapt itself to the pharyngeal wall and thus to find its way with the minimum of resistance. With the point always trailing, no trauma to the pharyngeal walls should occur.

Point up. In this position, it is folly to conclude that direct traction, unaided by laryngoscopy, is safe, even when the keeper is easily visible on inspection by direct depression of the tongue. The possibility that such traction will drive the pointed shaft into the pharyngeal wall must never be lost sight of, particularly traction applied without the aid of the laryngoscope and by the simple expedient of grasping the keeper with a curved hemostat.

There are in general two safe and feasible endoscopic methods applicable for pins in the hypopharynx with the point up. In the first procedure (Fig. 2), the keeper, usually the first portion of the pin seen on direct inspection with the laryngoscope or small esophageal speculum, is grasped through the central lumen with the toggling type of forceps, great care being taken that the teeth of the forceps meet within the eye of the keeper and do not grasp either the distal or proximal margins. Gentle traction is then made, allowing the pin to rotate to the side opposite the point until the pointed shaft occupies a transverse position across the mouth of the hypopharynx. This location tends to occur because the point remains in contact with the hypopharyngeal wall and so acts as a pivot on which, because of the toggling action, the remainder of the pin turns. So long as the length of the pointed shaft does not exceed the width of that portion of the hypopharynx in which it lies, the pin may then be withdrawn without risk of further penetration of the pharyngeal wall by the point. No pressure should be made by the exposing laryngo-

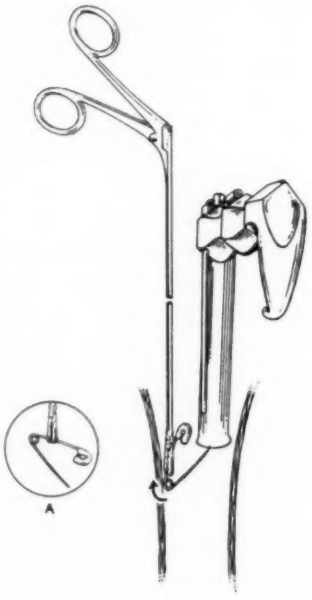

FIGURE 3. *Method of Extraction from the Hypopharynx. The lower end of the keeper shaft is grasped; this allows the pin to rotate. The tip of the laryngoscope guards the point. A — position of pin after "version."*

scope against any portion of the pin that must remain free to assume any position it seeks.

A modification of this method is applicable to pins in the hypopharynx that are of more than average size when it is believed that the pointed shaft might have difficulty in passing out through the pharyngeal diameter or when the pointed shaft is found deeply embedded in the mucosa or musculature of the pharyngeal wall. The shaft of the keeper is exposed to direct vision as far down as possible, preferably even as far as the spring (Fig. 3). An alligator forceps is then introduced parallel to and beside the laryngoscope,

but not passing through the lumen lest it obstruct the view of the exposed shaft. The latter is then grasped as close to the spring as possible and traction made laterally and upward, to displace the spring to the side of the hypopharynx away from the pointed shaft. At the same time, an effort is made to advance the lip of the laryngoscope in close proximity to the pointed shaft, and to hold it in contact with the latter as near the point as its engagement in the mucosa will permit. Thus, the lip of the laryngoscope tends to act as a fulcrum and also as a hindrance to any further advance of the point. With lateral and upward traction with the forceps, the perfect, if possible, a reasonably satisfactory method and so to practice it in the laboratory as to render it dependable except, perhaps, on the rarest occasions.

For the last fifteen years, it has been my practice with pins in this region to make use wherever possible of the Mosher safety-pin closer. The original model of this highly ingenious instrument has well stood the test of time and in my hands is easier to manipulate than the later and presumably improved variations. Barring minor changes in the obliquity of the distal end and lighting equipment, I can see no reason to alter it. Its use depends on the principle of ad-

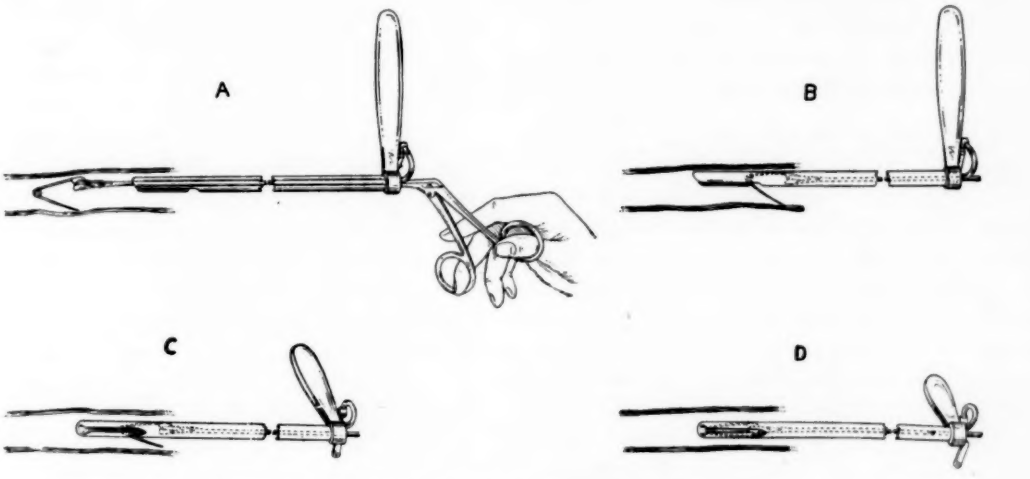

FIGURE 4. *Method of Extraction from the Midesophagus.*

A — the pin is grasped with toggle forceps passed through a Mosher pin closer; B — the Mosher closer is passed down over the keeper shaft until the spring reaches the transverse slot; C — the closer is rotated preparatory to pushing the pin downward with the pointed shaft impinging on the transverse slot; D — the pin is enclosed in the keeper and held by forceps ready to be withdrawn with the closer and forceps as a unit.

pin swings through an arc, the center of which is the edge of the laryngoscope. Thus is effected what Jackson[3] terms a "version" of the pin to the degree that the previously advancing point becomes converted into a trailing one, permitting ultimate safe removal by the spring or a point on the keeper shaft close to it (Fig. 3*A*). Once this method of fixation and traction is initiated, there must be no change in the relation of the laryngoscope or forceps lest this distort the mechanical principle and result in possible trauma.

Pins in the Cervical and Thoracic Esophagus

It is in the removal of open safety pins from this region that one encounters the greatest variability in technic employed by different operators and the expression of amazing ingenuity in the invention of specialized instruments. It is particularly for this reason that it seems wise to vancing the lower end of the instrument over the keeper and keeper shaft of the pin, while the extended and angulated pointed shaft remains outside and slides through a narrow slot extending somewhat over 2.5 cm. from the distal end of the tube. The proximal end of this longitudinal slot joins a wider triangular-shaped and much shorter lateral slot. Since the keeper of the pin has first been securely grasped with forceps applied under direct vision through the tube and held fixed in this position, without the slightest traction, there is no tendency for the pin to move (Fig. 4*A* and *B*). As soon as the point of union of the longitudinal and transverse slot reaches the spring, further advance of the tube is stopped, and the entire tube — but not the forceps — is so rotated as to bring the transverse slot in contact with the pointed shaft of the pin (Fig. 4*C*). With the tube fixed in this new position, the forceps,

still grasping the keeper, is advanced, forcing the latter downward into the distal portion of the tube and at the same time pushing the pointed arm of the pin against the lower margin of the transverse slot. This results in forcing the pointed arm into the tube until, with further advance of the forceps, the entire pointed arm is sheathed within the tube and the point thus completely protected (Fig. 4*D*). It is in no way essential that this closure result in the actual entrance of the point within the lips of the keeper of the pin itself. So long as none of the point extends beyond the tube, it is sufficiently protected. The entire system — pin, forceps and tube — in this new relation may then safely be withdrawn through the mouth.

The following fundamental precautions must be rigidly observed if this method is to succeed:

The diameter of the tube or Mosher esophagoscope must be adequate to admit freely the keeper of the pin. Only if this is possible can the maneuver be executed. For this reason, it is of paramount importance to determine the size of the pin with which one has to deal. It is desirable but not imperative that the blades of the forceps permit toggling of the keeper, since it is thereby somewhat easier for the pointed shaft to find its way into the longitudinal slot.

Under no circumstances, once the keeper is thus secured, should any traction be made with the forceps. Unconsciously to make such traction while the esophagoscope is being advanced over the keeper is to risk perforation or penetration of the esophageal wall by the pointed shaft, a far more serious accident with pins in the esophagus than with those in the hypopharynx.

Care must be taken in the final advance of the forceps that the pin is not pushed so far down toward the distal end of the tube that the pointed shaft is permitted to escape from the end. In this event, all the work must be repeated. The moment that the spring appears beyond the tube end, further forceps advance must stop, since the point will already be adequately sheathed. It is in the method of determining the limit of the forceps' advance that one comes to the final and, in many ways, most important requisite, the fluoroscope. I am convinced that fluoroscopic control is absolutely essential in the execution of this method of safety-pin removal from any portion of the esophagus below the cricopharyngeus muscle. Sheathing can undoubtedly be accomplished without this aid, but the added safety is a factor that, I believe, goes far toward assuring success. The best calculated and positive manipulation in the esophagus can sometimes go awry, and there is no better way to avoid such mischances than the utilization of the help from a competent radiologist, who can observe every technical step in the removal of an open safety pin by this method. At the Bronchoscopic Clinic of the Massachusetts Eye and Ear Infirmary, it is an inflexible rule that all such and many other esophageal and bronchial foreign bodies be removed under fluoroscopic guidance.

Even under the most favorable circumstances and with every possible help, it may not always prove feasible to adhere to the determination to remove an open safety pin through the mouth, even though it is admitted that it entered by this route. If, for any reason, the sheathing method does not proceed to a successful termination, it may be advisable to advance the pin to the stomach and there deposit it. This involves putting one's pride in one's pocket and a chagrining although perhaps only temporary loss of face. A foreign body in the stomach and an untraumatized esophagus are far better for the patient than peroral removal and subsequent mediastinitis. It is the opinion of most general surgeons that an open safety pin in the stomach, if not of undue size, will pass harmlessly through the remainder of the digestive tract without the necessity of any surgical intervention. Unquestionably, it may at times be necessary to remove from the stomach an open pin that, after a reasonable delay, refuses to pass through the pylorus, but this circumstance in my experience has been rare. Such pins in the stomach will not infrequently be found in the stool within twenty-four to forty-eight hours.

For those who dislike such deposition of the pin in the stomach, there is available the method of gastric version in which the pin, grasped by the spring from above, is carried into the stomach, allowed to reverse its position and then brought out, point trailing, through the mouth. With this method, I have had no clinical experience.

The foregoing description of sheathing an open safety pin applies quite obviously to those cases in which the pin rests point up. With the point downward in the lower esophagus, the problem is simpler, involving exposure of the spring by means of the esophagoscope, the grasping of the spring with forceps, preferably toggling, and the withdrawal, at least to a partial extent, of the spring into the lumen of the tube. Here again, although not indispensable, fluoroscopic guidance may be more valuable than one might at first suppose.

Pins in the Respiratory Tract

The larynx. As already explained, an open safety or clasp pin lodges oftenest in the larynx, point up. If the point itself is clearly visible above the ends of the vocal cords in the anterior commissure, it is usually not difficult to grasp the keeper with alligator forceps passed through the laryngoscope and to slip the pin upward without engaging the point in the soft tissues. If, however, the point of the pin is engaged in the anterior glottic wall below the point of approximation of the vocal cords, simple traction will be unsuccessful. Under these conditions, I have found the method of partial straightening of the pin through the larynx a safe and satisfactory one. With the larynx exposed by the laryngoscope, the presenting keeper is grasped with toggle forceps, the keeper lying, as already explained, in the sagittal plane (Fig. 5*A*). The tip of the laryngoscope is then held firmly near the base of the epiglottis and presumably just above the pointed end of the pin (Fig. 5*B*). With the laryngoscope thus fixed, traction is made on the keeper, which is then withdrawn through the lumen of the laryngoscope, in the process of which the normal acute angulation of the shafts is expanded into a wide, obtuse angle or almost into a straight line (Fig. 5*C*). This principle presupposes that the point of the pin will not advance beyond the lip of the laryngoscope, so long as the latter is held rigidly in place, until the pin is straightened out. There is sufficient laxity of the interarytenoid space to permit the spring to rotate around the axis fixed by the laryngoscope. Even were the point to advance slightly beyond the lip of the laryngoscope, it is unlikely that any serious trauma would be inflicted in the region of the anterior commissure. With the point facing posteriorly, one might hesitate to execute

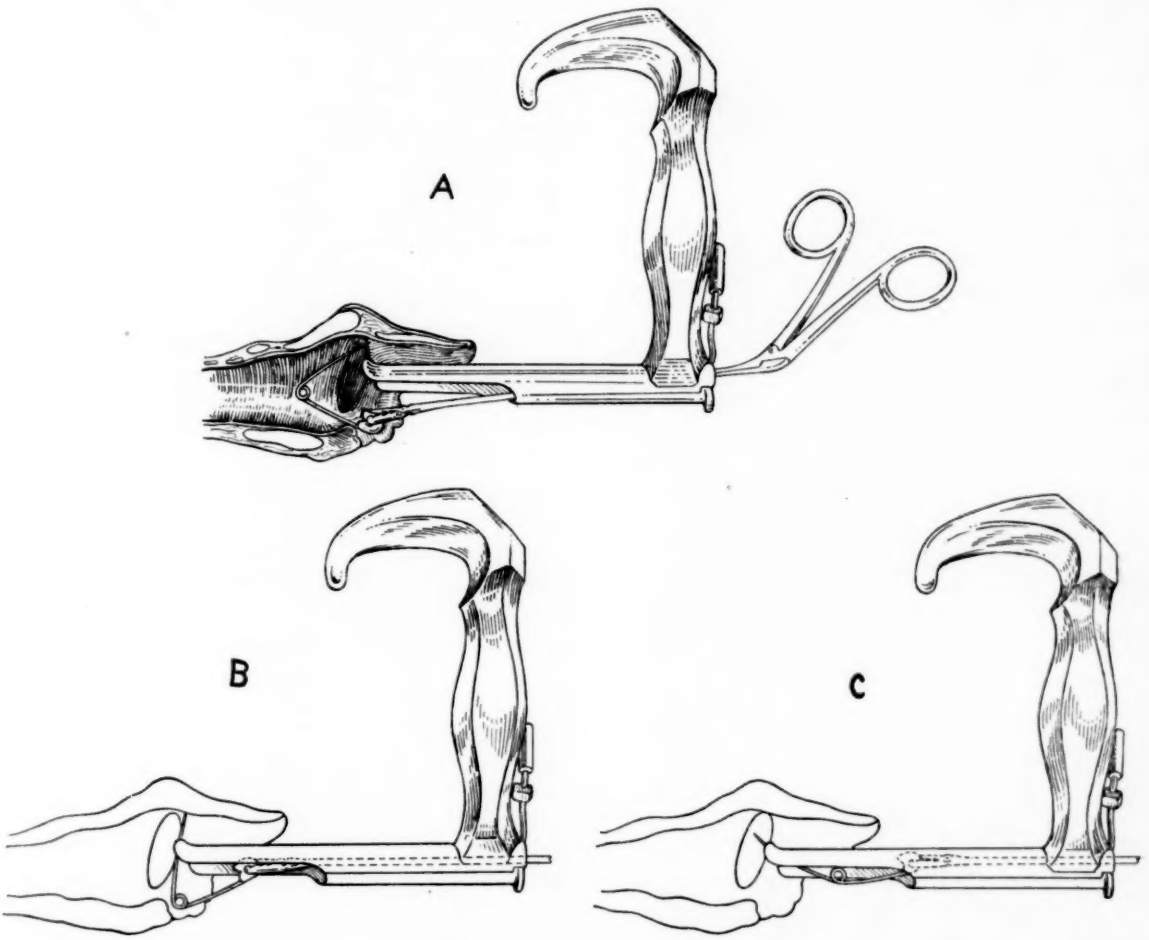

FIGURE 5. *Method of Extraction from the Larynx.*

A — *the keeper of the pin is grasped by toggle forceps, while the tip of the laryngoscope protects the point;* B — *traction on the keeper is made while the laryngoscope prevents advance of the point;* C — *the straightened pin is withdrawn through the laryngoscope, with point trailing.*

this maneuver, lest the party wall between the larynx and esophagus be perforated, but fortunately this accident is extremely uncommon.

The bronchus. In the event that an open safety pin of relatively small size with a necessarily less than ordinary spread passes through the larynx, it may come to rest in a main bronchus, oftenest just below the bifurcation of the trachea and usually with the point up. Such a pin could, of course, conceivably lodge in the trachea, but if of sufficiently small size to pass the subglottic region, it is far likelier to descend and lodge in one or the other of the major bronchi. In my very limited experience with this situation, I have found the straightening method, somewhat as above described with reference to the larynx, to be satisfactory. It is first of all necessary to pass the bronchoscope into the invaded bronchus and visualize the keeper of the pin. The bronchoscope must be of sufficient caliber to permit, in much the same manner as the Mosher esophagoscope, the entrance of the keeper into its lumen. There is no question, however, of closure, since it is impossible, owing to the decreasing caliber of the bronchus, to advance the bronchoscope beyond the pin (Fig. 6*A*). The keeper is grasped with toggle forceps, and the bronchoscope slipped downward until the spring impinges on the more distal edge of the bronchoscope (Fig. 6*B*). The latter is then held firmly in place, and steady traction is made on the keeper, thus straightening the pin into the bronchoscope (Fig. 6*C*). The apparent risk of this procedure is greatly minimized by the fact that such bronchial pins usually lie with the point just proximal to the carina. As traction is made against the lip of the bronchoscope, the point tends to rotate across the carina and even into the orifice of the opposite main bronchus, thus affording it a temporary resting place until it is converted into a trailing point. As already stressed, it is of the greatest importance to know beforehand the size of the keeper and to utilize a bronchoscope the lumen of which is assuredly large enough to admit the keeper freely. In an infant, this necessity may provoke complications, since a sufficiently large bronchoscope may pass the larynx with difficulty and, later, resultant laryngeal edema may require tracheotomy. This, however, is not serious provided that the pin has been successfully

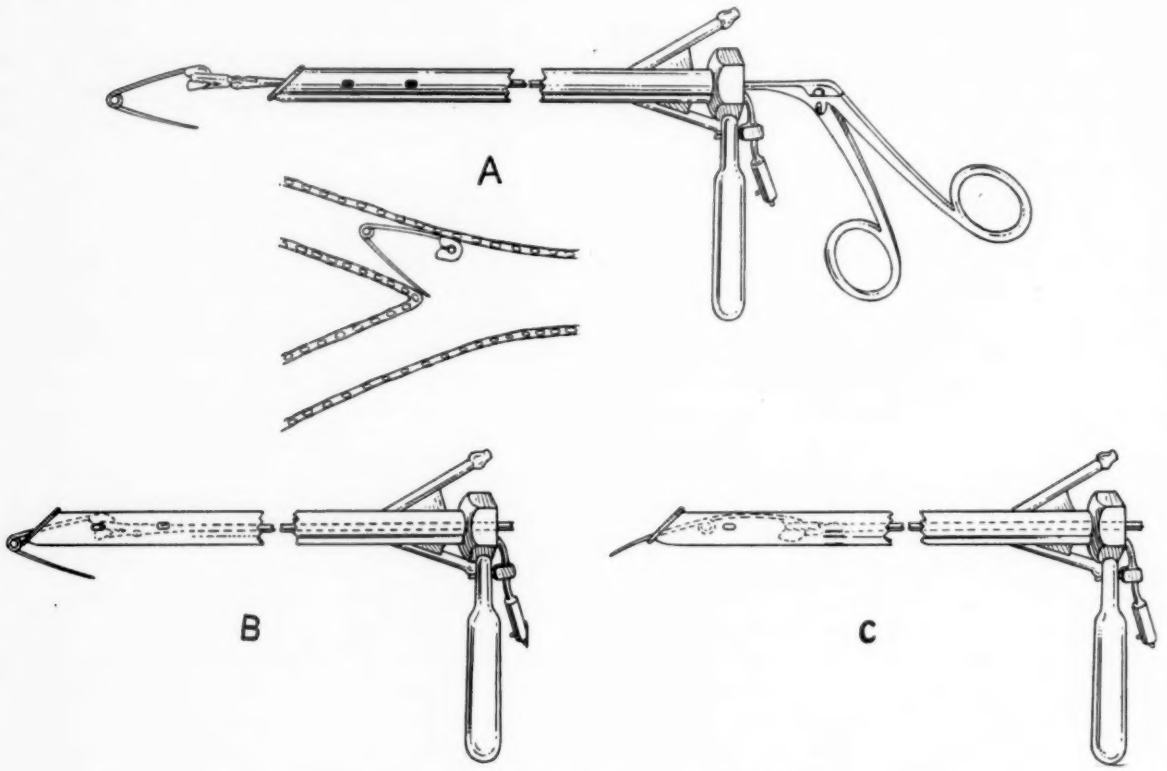

FIGURE 6. *Method of Extraction from the Bronchus.*

A — *the pin is grasped with toggle forceps, preparatory to advance of the bronchoscope over the keeper;* B — *the keeper is drawn into the tube, with the lip of bronchoscope braced against the spring preparatory to straightening into the tube;* C — *the pin is straightened into the bronchoscope, with point trailing.*

removed without trauma to the tracheobronchial tree. Fluoroscopic assistance in such a situation is not so essential as it is with pins in the esophagus, but may be of distinct assistance to the bronchoscopist.

Complications

The complication most to be feared in connection with an open safety or clasp pin in the food passages is perforation of the esophagus. The wide and unyielding spread of the spring seems offhand to make this a likely occurrence in any case in which the pin is not removed immediately. Rather strangely, such preoperative perforations are quite rare, and open pins have been known to remain in the esophagus for weeks or months without perforation or serious local infection. The considerable mobility of the esophageal wall doubtless accounts for this fortunate circumstance. Hence responsibility for the definite development of alarming symptoms of perforation or mediastinal infection following attempted removal must usually be placed squarely on the shoulders of the operator, who can feel scant solace in the thought that the pin may have perforated spontaneously. Postoperative elevation of temperature, painful deglutition, a rising leukocyte count and definite roentgenographic evidence of an increasing prevertebral swelling with or without air in the tissues —any or a group of these symptoms strongly suggest the need for prompt consultation with a thoracic surgeon, with strong probability that external drainage of the upper or lower mediastinum will be advisable. More detailed consideration of the indications for such surgery does not fall within the scope of this paper. Suffice it to say that impending tragedies have been averted by prompt resort to external drainage of an esophageal perforation.

Conclusions

The open safety pin is a relatively common foreign body in the upper air and food passages. Early diagnosis is best assured by x-ray examination of any patient in whom such a foreign body is suspected.

Technics of removal vary with the location of the pin, but familiarity with one dependable method is more valuable than a theoretical knowledge of several. Fluoroscopic aid is indispensable in the removal of pins in the cervical and thoracic esophagus, and is a distinct aid with those in the tracheobronchial tree.

Evidence of a complicating esophageal perforation demands prompt consultation with a thoracic surgeon.

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ENLARGEMENT OF THE HEART*

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THERE has been a great deal of talk about congestive heart failure during the last few years — indeed, to the extent that it has taken the limelight of medical interest, so far as the heart is concerned. Important as it is, however, it is far less vital and should receive less attention by the medical profession than two other cardiac problems. The relatively greater significance of the first of these, the causes of heart damage or strain, is readily and widely admitted, but the second is often neglected and indeed sometimes forgotten, namely, heart size — whether or not there are any other abnormalities, such as symptoms, murmurs and arrhythmias.

Two hundred and sixty-two years ago, Théophile Bonet published a fundamental collection of autopsy reports, the *Sepulchretum*, which was critically reviewed and amplified in a sort of third edition by Morgagni nearly a century later, under the title *De Sedibus et Causis Morborum*. In the original edition in 1679, there were a number of cases showing enlargement of the heart; one of these patients had suffered from shortness of breath that before death had been routinely ascribed to pulmonary disease, which was not found post mortem. The association of a serious symptom with a large heart was spoken of with surprise by the observers of that case and was evidently the beginning of the realization of the importance of cardiac enlargement. A few decades later, in 1728, there was published a book, in large part on this subject, written by Lancisi, physician to Pope Clement XI; it was entitled *De Motu Cordis* [following the post-Harvey tradition] *et Aneurysmatibus*, which meant large hearts as well as large aortas.

Thus, quite rightly, the first cardiac abnormality

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of consequence to be described and studied in pathology and clinical medicine was enlargement of the heart. With the advent of auscultation, however, medical interest was turned away from heart size, on which percussion, already introduced, was unable to focus attention, and concentrated for a century or more on heart murmurs, which were too often interpreted as evidence of valvular disease. The textbooks for many decades were filled with chapters on every possible valve defect, only a few persons, like Mackenzie, trying to redirect attention to other cardiac findings, after the turn of the century. At last, cardiologists are approaching a saner and wiser viewpoint, although I dare say they are still simply in the middle of the process of proper cardiac orientation. It is this revolution and evolution in cardiovascular knowledge that has rendered a cardiologic vocation absorbing and satisfying for those who were so fortunate as to enter that field two or three decades ago.

The first of the problems of heart size is the most difficult of all, namely, the range of the normal size. It has been left to the last to study, for, fundamental though it is, it has not appealed to the pioneers who have been unearthing, with almost every spadeful, nuggets of interesting, spectacular and unusual pathologic conditions of the heart. I have been as guilty as the next man in this respect, witness to which is the record that my laboratory still holds in medical literature of enormous heart volume — seven times the normal. Now at last, except for a few who have patiently tilled the field for some years, cardiologists are turning to the more vital problem of the determination of the beginning of cardiac enlargement in any given case.

What is the normal heart size, and how can it be ascertained in a particular person? Even the anatomist, pathologist and anthropologist cannot limit the heart weight and volume for the normal person within a narrow range. Roughly, the weight of the heart has been considered normally to average 0.5 per cent of the body weight, but this is a very imperfect estimate indeed. Body build and other hereditary manifestations that have not yet been worked out are undoubtedly important correlative factors; the aid of anthropologic data is needed to secure greater accuracy of fundamental measurements, and this aid is only now beginning to be sought.

With this difficult enough problem to begin with, one now must face the additional difficulty of the clinical determination of heart size. Three methods are available. First and simplest and often quite accurate is the position of the cardiac apex impulse by palpation. Sometimes this is easily determined, sometimes only with difficulty, and sometimes it is impossible. In those cases in which the maximal apex impulse is found in the fifth intercostal space within or on the left midclavicular line, one can be quite sure that there is no significant cardiac enlargement. In such cases, it is still essential to percuss the position of the left upper border in the third and fourth interspaces, — and this can be done in most cases with reasonable accuracy, — to determine if there is undue prominence of that part of the heart, a finding common in cases of mitral stenosis or cor pulmonale with an enlarged right ventricle but with the apex impulse not far from the normal position.

Next, a word is due about percussion. Inaccurate and disappointing as this method of examination may be in many cases, in comparison to x-ray study, it is often a useful procedure in many patients when x-ray examination is impracticable or not immediately possible. There are many such cases, and if one sits back with folded hands complaining of the inferiority of percussion to the x-ray film, one will miss valuable evidence of heart size that may be obtained only by percussion when the apex impulse is felt with difficulty, or not at all, and when x-ray examination is not available. It is necessary to acquire skill in percussion, which cannot be learned overnight. Most of those who scorn percussion do not want to bother to learn the technic or through long neglect have lost all confidence in it. Even though I fluoroscope each one of my patients in my office, I percuss the chest first and thereby control my percussion findings and keep up a certain practice that proves helpful when I encounter patients elsewhere, as at home. I can recommend this procedure.

Too much confidence about heart size, on the other hand, is often yielded to an x-ray report, — not that the x-ray picture per se is not correct, although even its accurate transcription and original interpretation need a check, for which more than once physical examination has served me well, — but deductions from measurements of the x-ray heart shadow must be made in borderline cases only with the greatest care. Here, too, there are wide limits of the normal, even for the same height and weight and age — so wide in fact that a good many competent and experienced roentgenologists do not bother even to make any measurements unless requested. Other roentgenologists furnish a long list of measurements of diameters, arcs, area and even volume; these are often interesting but far less valuable than an actual view of the x-ray film itself, or best of all, of the subject

in the flesh under the fluoroscope. There are too many imponderables for one to have confidence in a few ponderables, if overmuch is made of them. My own attitude is a compromise between these two extreme points of view. I favor those who scoff at emphasis on detailed measurements, and yet, poor as they may be, the measurements to my mind are better than none, for x-ray films may be lost or difficult to secure at any given moment for detailed study or comparison with previous records; and figures, rightly or wrongly, mean more to me than mere statements of normal heart size or of slight, moderate or great enlargement.

Just a brief word, before leaving x-ray heart study, about the best measurements to use. Combinations and correlations of the various diameters have been introduced, and the heart volume has also been roughly measured, but none of these procedures are practicable for routine use, because of various difficulties of technic or of wide scatter of the normal range. Three methods in common use are equally applicable to orthodiagraphy and to seven-foot films, and are satisfactory in their routine employment, when one remembers that the heart shadow is larger by nearly 10 per cent in the seven-foot film than in the orthodiagram. The commonest method, but one that should be discarded because its range of the normal is the widest, is the so-called "cardiothoracic ratio," that is, the division of the transverse diameter of the heart by the internal transverse diameter of the thorax; here the normal range is tremendous, from 33 to 55 per cent. The other two methods, both open to objection but the best for routine use, are the correlation of the transverse diameter of the heart shadow with height, weight and age, according to the Hodges-Eyster formula, nomogram or slide rule, and the correlation of the area according to height, weight and age. There are special advocates of both, and since the measurement of the transverse diameter is much simpler and quicker technically, I am at present using it with the help of a simple slide rule. It is as quick as and better than the cardiothoracic ratio, which I have recently discarded. Actually, however, for reliance one needs something better than anything as yet available.

The most awkward dilemma is an inability in many cases to detect the presence of slight cardiac enlargement, an early sign of serious heart strain and therefore an essential thing to know in any cardiac patient. The range of normal is so wide that any case may change from a small normal heart size to a large normal heart size without one's realization of the fact unless one has early careful measurements of that particular heart to begin with.

Formerly, cardiac enlargement was regarded as evidence that the heart was strong and compensating for whatever strain was present, but it has gradually become realized that a large heart is a bad sign and evidence that the myocardium is not a normal healthy muscle and that it is no longer able to resist the strain. It is an indication that more and more enlargement is likely to follow, resulting in myocardial failure of the congestive type, unless the strain can be eliminated. In fact, any cardiac enlargement is already evidence of some failure of myocardial strength, although not at first of the grade to result in congestion. Slight and moderate grades of increase in heart size may persist with little change for years, however, and are not to be viewed with immediate apprehension. Nevertheless, they are not good signs, and important trouble threatens if marked enlargement is found or if the size steadily increases under observation for a relatively short time.

Enlargement of the heart may be due to acute dilatation, to preponderant hypertrophy or to both hypertrophy and dilatation of comparable degree. The best theory of its onset is that it starts as an overstretching or dilatation of the myocardium and that work hypertrophy is in large part a reaction of the heart muscle to injury and a failure of the myocardium to return to its original size. Little by little, this stretching in many cases goes on until the heart becomes very heavy, although often with relatively little dilatation of the heart chambers, and the process accelerates to a state of clinical myocardial insufficiency from which congestive heart failure results.

There has been a return to the old-time diagnosis of acute dilatation of the heart, once applied indiscriminately and often to the wrong patients, then two or three decades ago abandoned disdainfully, but now acknowledged as a correct appellation for certain cases. Four conditions are occasionally responsible for a fairly acute dilatation of the heart, two of which — namely, severe acute rheumatic myocarditis and excessive heart rate in paroxysmal tachycardia — affect both ventricles; the third, acute massive, myocardial infarction, involves the left ventricle primarily, and the fourth, massive pulmonary embolism, involves the right ventricle (the acute cor pulmonale). The acute dilatation may precipitate congestive failure and death, it may clear up almost or apparently completely on subsidence of the acute strain, or it may, as in many cases of acute rheumatic and acute coronary heart disease, leave permanent enlargement of the heart, consisting in both dilatation and secondary hypertrophy, which may in chronic rheumatic heart

disease be ascribed wrongly, in toto at least, to coincident valvular deformity.

Some hearts, especially those under the strain of systemic or pulmonary hypertension and of aortic stenosis, show at first and for years preponderant hypertrophy, dilating appreciably only at the time of final failure. Others, especially those with free aortic regurgitation, show much dilatation at an early stage and also the highest degrees of hypertrophy; the heaviest and often the most voluminous hearts belong in this group and have been labeled "cor bovinum." The record-breaking hearts in volume, however, are those with mitral valve disease and auricular fibrillation in which the left auricle and often the right ventricle and right auricle become enormous, whereas the left ventricle is normal or actually small.

In most cases of cardiac enlargement, the strain is left-sided, from systemic hypertension, aortic valve disease or myocardial infarction, and the left ventricle consequently enlarges first and sometimes is the only heart chamber to do so. But very frequently it becomes incompetent, and then, in order, first the left auricle, next the right ventricle and finally the right auricle become enlarged. It is enlargement of the left ventricle that increases the heart weight preponderantly; that of the left auricle displaces the greatest space.

The right ventricle is far oftener enlarged than cardiologists formerly realized, chiefly because attention has been distracted by the much more obvious enlargement of the left ventricle, and because the right ventricle has been neglected, even by the pathologist. Another reason is that when it is enlarged alone, with little or no dilatation, it may escape notice clinically even on the most careful x-ray examination, as happened in a case of extensive pulmonary emphysema recently considered at a clinicopathological conference at the Massachusetts General Hospital, in which at autopsy the right ventricular wall measured 7 mm. in thickness (normal, 3 to 4 mm.); it is quite probable, however, that an electrocardiogram would have afforded the only clue, but an essential one, in the form of a considerable degree of right-axis deviation. Finally, it should be remembered that the commonest cause of enlargement of the right ventricle is failure of the left.

Many different factors may cause cardiac enlargement, some extracardiac, like anemia and myxedema, and some intracardiac, like rheumatic myocarditis, myocardial infarction and valvular disease. In either event, the strain may be largely acute and temporary, and the enlargement may subside; but also in either event, the strain may persist and the enlargement may become permanent. Chronic enlargement is, of course, most commonly due to intracardiac disease and systemic hypertension. Quite often, particularly in older patients, more than one factor may be active at the same time. One condition, chronic pericarditis, was once wrongly blamed as a cause of great cardiac enlargement; a pericardial effusion can of course simulate a large heart, but it really acts like occasional chronic pericarditis to constrict the heart and so to make it smaller.

The prognosis of cardiac enlargement is quickly and fairly well summarized in the observation that "the larger the heart, the worse the future." Although there are exceptions, this rule holds if other things are equal.

Finally, the treatment of cardiac enlargement is twofold: first, relief of the underlying strain, which is not always possible directly, but which one can often help a great deal by reducing other strains of daily life; and secondly, the optional routine use of a daily ration of digitalis to help to prevent or retard further cardiac enlargement and failure. It seems reasonable to give 1 or 1½ gr. of digitalis daily for this purpose; the value of this drug has not yet been proved, and it will be difficult to prove.

* * *

Cardiac enlargement should be suspected in patients whose hearts are under strain, even though they are free of symptoms; it is possible to do more good at an early stage than after heart failure has set in. On the other hand, it is equally important not to establish cardiac neuroses thereby, and especially not to mistake for cardiac enlargement a heart size that is on the borderline of normal unless undue heart strain is or has been present.

NUTRITION IN WAR*

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DURING recent years, this nation has unquestionably been living on a dietary borderline in ability to supply the nutritional factors necessary for physiologic health. Deficiency disease is endemic in this best-fed nation because more than 40 per cent of the families do not have the money to buy food for an adequate diet, because those who have the money do not have the knowledge, interest or diet habits to buy the proper foods, and because foods have been refined, processed and canned without regard to the effects on nutritional factors. As times goes on, it is being demonstrated experimentally, clinically and in the daily life of the people that the majority are knowingly or unknowingly ailing from the insidious effects of deficiency disease.

Perhaps the most important cause of these deficiencies is a too-strict adherence to the precept "Cleanliness is next to Godliness," for Americans have insisted that their foods be whitened, polished and refined. In removing the color from foods, they have unconsciously destroyed the precious compounds on which, as an evolving race, they developed a dependence.

Eight decades ago, the roller-mill process for flour manufacture was invented, and today all white flour is made by that process. Jolliffe[1] has calculated that, to obtain the thiamin lost by consuming this white flour instead of the stone-milled flour of one hundred years ago, one would have to consume each day, *in addition to the ordinary diet*, about 1½ pounds of fruit, 1⅓ pounds of potatoes, 2 pounds of other vegetables and 1⅓ quarts of milk. Because it is difficult to increase one's food intake by so large an amount, and because such a procedure would be expensive, population groups with low incomes have suffered most from the refinement of the dietary and show the highest incidence of deficiency disease. The poor are of prime importance in considering measures to eliminate this condition, for those in higher economic brackets can more or less successfully, but not with certainty, make up for the losses caused by refinement by consuming more fresh vegetables and fruit.

The amount of bread consumed by a community is usually in direct proportion to its economic status, for bread is the food of the poor. It was fundamentally sound, therefore, when the Committee on Food and Nutrition of the National Research Council was organized last fall, that its first action[2] was to decide the vitamin and mineral potencies that bread and flour should have to make these staple foods, which together constitute one fourth of the average calorie intake, carry their rightful nutritional quotas. Bread will become a staff of life on which man can again lean for true nourishment.

It is expected that cereal food products will be similarly enriched, and that the slight cost will be absorbed by the manufacturers. It is hoped, also, that the Committee on Food and Nutrition will continue to catalyze improvements in the basic foods of the poor, especially sugar and fats, and to encourage the consumer to purchase those inexpensive foods that will give him the most for his food dollar.

The problem confronting this committee is to see to it that the foods most readily available, the staple foods, are so nutritious and still so inexpensive that it would be exceedingly difficult for a person with little knowledge of nutrition to select a diet that would be deficient over a reasonable period.

Why is this especially significant at this time? Because an ill-fed nation is a sick nation.

Drummond and his group,[3] for example, made a comparative study of the life history of two groups of rats, one of which (483 animals) was maintained on what they called an "adequate" diet and the other (556 animals) was fed a diet providing a suboptimal intake of the vitamin B complex, the chief defect being that of vitamin B_1. The life span was shortened, reproduction was impaired, and the incidence of gastrointestinal lesions was definitely increased (Table 1).

In similar experiments, McCarrison[4] apparently used a more adequate diet than Drummond, for he noted no gastrointestinal disorders in rats, rabbits, dogs and monkeys fed on this diet, whereas animals on a diet deficient in minerals, vitamins and proteins, and identical to the dietaries of a large mass of people, showed some gastrointestinal or organic disorder, usually several. Numerous other experimental and clinical results clearly indicate the great value of good nutrition and lead one to ask whether many of the maladies of to-

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day are the result of malnutrition and undernutrition.

At the beginning of this century, it was thought that nutrition consisted in supplying calories for fuel, protein for muscle formation, water and a few minerals, such as calcium, phosphorus, iron and iodine. Foods were analyzed on that basis, and diets were formulated in terms of calories.

TABLE 1. *Effect of Subsistence on Diet Deficient in Vitamin B Complex.*[3]

GASTROINTESTINAL LESIONS	ADEQUATE DIET (483 ANIMALS)	INADEQUATE DIET (556 ANIMALS)
Dilated stomach	9	60
Gastric erosion	27	88
Chronic gastric ulcer	27	80
Perforated ulcer	1	7
Hair balls	8	44
Distended intestine	22	52
Intestinal ulcer	4	17
Cecal ulcer	8	42
Perforated ulcer	2	4

TABLE 2. *Nutritionally Essential Factors.*

CALORIES (carbohydrate and fat)			
PROTEINS (aminoacids)			
Phenylalanine	Methionine	Histidine	Leucine
Tryptophane	Isoleucine	Arginine	Lysine
Threonine			Valine
VITAMINS			
Vitamin A	Pyridoxine (vitamin B_6)		Vitamin D
Thiamin (vitamin B_1)	Biotin (vitamin H)*		Tocopherol (vitamin E)
Riboflavin (vitamin B_2)	Para-amino benzoic acid*		Vitamin K
Nicotinic acid	Ascorbic acid (vitamin C)		Choline
Inositol*			Linoleic acid
Pantothenic acid			
MINERAL ELEMENTS			
Calcium	Sodium	Chlorine	Zinc
Phosphorus	Potassium	Sulfur	Iodine
Iron	Magnesium	Copper	Cobalt
	Manganese		
WATER			

*Evidence of human needs is fragmentary.

It is now realized that nutrition is much more involved, and that more than three dozen substances and elements are required for normal physiologic functioning (Table 2). Each of these substances must be supplied in adequate amounts else deficiency disorders will result.

Nutrition of Armed Forces

Since the majority of the American people have minor nutritional ailments and a very substantial minority are definitely deficient in several nutritional factors, a good proportion of the men entering military service are undernourished. They are being given nutritionally balanced diets definitely superior to those on which they lived at home. One can therefore expect an improvement in the health, vigor and mental and physical stamina of these men. Military authorities are keenly aware of the importance of sound nutrition in preserving the morale of the men, and they are constantly being advised by the best medical and nutritional experts in an effort to make the American soldier and sailor the best fed in the world.

The value of good nutrition to a nation at war is shown in the recent reports by Williams and his co-workers,[5, 6] who found, for example, that, in an experimental group, a diet deficient in thiamin produced moodiness, sluggishness, indifference, fear, depressed mental states, mental fatigue, dizziness, backache, insomnia, anorexia and physical fatigue. Morale and stamina cannot be expected of thiamin-hungry troops or populations.

The German high command has long studied the problem and has made painstaking preparations lest the present war be finally lost owing to the malnutrition of the German troops and people. In World War I, malnutrition was a powerful factor in leading the Central Powers to seek an armistice. The food blockade of that war was so effective that starvation and its accompanying deficiency diseases were rampant in the general population. The advances that have been made in nutritional science since 1918 have been so great as to make it almost impossible to blockade the Nazi nation. All the important vitamins man needs can now be synthesized within Germany at little cost.

TABLE 3. *Cost of Important Vitamins Required by an Adult.*

VITAMIN	DAILY OPTIMUM	COST PER DAY
A	5000 int. units	$0.0008
B_1 (thiamin)	2 mg.	0.0013
B_2 (riboflavin)	3 mg.	0.0030
Nicotinic acid	20 mg.	0.0008
C (ascorbic acid)	75 mg.	0.0053
D	400 int. units	0.0001
		$0.0113

Today, the American manufacturer of food or vitamin products can purchase these vitamins for so little that a daily optimum costs less than 1.2 cents (Table 3). If the Government were to supply a population of 50,000,000 with these needed vitamins, the basic cost would be only $206,000,000 yearly. Since these current prices in America are undoubtedly higher than those in Germany, it is evident that the Nazi command can easily supply sufficient vitamin supplements to protect the German armies and people against vitamin-deficiency disease, especially since a goodly fraction of the vitamin requirements can be contributed by rationed foods and ersatz.

Although vitamin starvation had much to do with the defeat of the Central Powers during World War I and would likely not be a factor in the defeat of the Axis in the present conflict,

protein starvation may be a significant factor. Calories can be obtained from many kinds of foods, vitamins can be synthesized in the chemical laboratories, minerals can if necessary be provided in the form of inexpensive tablets, but the protein requirements of a people cannot be met without good foods. The constituent aminoacids of proteins are needed for the formation and replacement of muscle tissue, and the special functions of many of these aminoacids cannot be performed by any other compound; only a few of these acids can be synthesized cheaply by the chemist.

A nation at war must have adequate supplies of good proteins if it is to survive. Therefore, foods rich in excellent protein (meat, milk powder and cheese) are today preferred for shipment to England.

The proteins of plant tissues are generally inferior to those of animal tissues because they are deficient in one or several of the essential aminoacids. It is, of course, possible to combine plant proteins in the dietary or in food mixtures so as to obtain adequate amounts of these aminoacids, but this requires special knowledge.

It has been authentically reported that the German forces, in garrisons and in active warfare, are living on dried foods and dried food mixtures. During the last ten years, the German government has fostered considerable research and developed methods for dehydrating foods without destroying nutritional factors and changing the taste. Without doubt, Germany stands far in advance of other nations in this vital field of food technology, which has not had the proper development in this country. The Nazi armies can be fed with tasty, nutritious, dehydrated foods, which require no refrigeration and are one fifth as bulky as ordinary foods. The rapid maneuvers and advances of the *Panzer* divisions in recent battles would have been difficult without this simple method of feeding.

Furthermore, in these rations, plant proteins rather than animal proteins have been used. It would be difficult to supply animal proteins during a long war, for the fodder needed by the livestock cannot be imported. For many years there has been an intensive planting of soybeans in the Balkans, in Bessarabia and in Germany. This interesting legume, long a staple food in the Orient, will grow on a variety of soils, requires little attention, and yields a food protein the important aminoacids of which approximate, in variety and proportion, the proteins of meat, milk and egg. Although considerable reliance is being placed on soybean protein, it is not certain that enough is being produced to meet Axis needs. But this would be of no consequence, since one can be sure that the Nazi command will allow protein starvation to appear in the conquered nations and use their foodstuffs in feeding the Nazi armies and the German population.

Although it is my belief that the Axis will not be starved out of this war, there is a grave possibility that England will be counterblockaded into starvation: protein starvation, vitamin starvation and, eventually, calorie starvation. Britain has long been an industrial nation, importing raw materials and exporting manufactured goods, meanwhile relying on importations to supply 40 per cent of her food. She has lost several neighboring islands from which important quantities of vegetables and dairy products have been received in the past. She could not import enough fodder for her pigs, poultry, sheep and beef cattle, and has slaughtered many.

However, Britain has increased the number of milk cows so that an unrestricted amount of milk will be available to nursing and expectant mothers, to young children and to hospitals. Over 2,000,000 acres of grassland have been plowed up in response to a bonus of £2 per acre and planted mostly with grains (barley, oats and wheat). British leaders have stressed the point that malnutrition can be avoided with sufficient milk, vegetables and potatoes, and that starvation can be prevented with sufficient bread, vitaminized fat, potatoes and oatmeal. Imports of fats, sugar, dried fruits, frozen meats, canned meats, canned tomatoes, milk powder and cheese have been preferred, for these foods have high-calorie content per ton of shipping space, contain fine proteins and critical vitamins, and supply the special needs of the British people.

Foods such as meat, fats, butter and sugar have been rationed along with many nonessential food items. Any war involves a marked simplification of the foods of the people, with an accompanying loss in palatability and richness and a limitation of choice. There is evidence of reduction in body weight in some sections of the population. So long as this restriction is not great, the rationed peoples may be well off, since they report that they feel better. Severe restriction would be serious since, even in normal times, one must consume more than 1900 calories as food daily to obtain needed vitamins. The best choice of foods supplies less than three quarters of the required daily vitamin needs, with the possible exception of ascorbic acid. Thus, adults have a choice between deficiency disease and gluttony. If one can judge from the appearance of the adult population, they are choosing gluttony — the better choice under the circumstances. Is there not some rela-

tion between the good nature of the fat man and his better nutrition?

Rationing of food is not new; in fact, it is an integral part of planned economy. It need not work hardship on a people or adversely affect its morale. The poor have always been rationed, for poverty limits their choice of foods. Furthermore, since the people of a warring country are now truly in the front lines, they must be ready to resign themselves to the restrictions and methods of the soldier, even to the extent of eating concentrated food mixtures.

It should be pointed out that concentrated mixtures consist of several foods, cooked separately, blended and mixed; they are not ersatz in any sense, since only water is removed. That is all that can be done, because one cannot concentrate the 3000 calories or the 100 grams of protein that an adult needs daily. Test-tube feeding or capsule feeding is the invention of the newswriter, for adult man cannot be fed on less than 1⅓ pounds of concentrated foods daily.

Feeding the Population in War

Research has shown that visual acuity, mental and physical health, morale and endurance may be impaired by nutritional deficiency. It is therefore imperative that workers connected with defense industries be well fed. It may be advisable to imitate England's soup kitchens by feeding the workers in these vital industries nutritious midday meals. Field tests have shown that the improvement of substandard diets in groups of workers results in increased output and a reduction in accidents.

One of the most important functions of the Government is the school-lunch program, which endeavors to feed needy children with surplus foods. In the present war, this program should be extended to include all undernourished children and to improve the lunches so that an adequate diet will be assured. The effect of a continued program on the health of these children and on their future usefulness to themselves and others cannot be predicted too optimistically. Clinical experiments have shown its advantages.

Some method will have to be devised to care for the remainder of the population who cannot afford an adequate diet. Until then, the admirable Food Stamp Plan should be extended. Certain foods now purchased under this plan may soon lose their status. In fact, it appears that wheat is the only true surplus, and efforts should be made to curtail its production. Soybean, peanuts and similar foods of superior nutritive value should be grown in its stead.

Postwar Problems

When the present conflict is over, there will be millions in various degrees of starvation who will need to be fed. There are millions today in this condition, but they cannot be reached. One can only hope that they will survive to the armistice. Hundreds of thousands will undoubtedly perish, the victims of disease resulting from their nutritional deficiencies. Those who survive may long carry the scars of their starvation, possibly into future generations. In looking ahead to the day when these unfortunate people may be fed, groups are anticipating the needs and devising a program to fill them.

* * *

This is a critical and unhappy era. We can properly feed our own people and assist in the feeding of our valiant democratic allies. Britain must not be starved into submission. When the war ends, it will be our duty to help in the feeding of unfortunate peoples made destitute by war. This is the challenge on the food front — and we must meet it.

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MEDICAL PROGRESS

ARTERITIS: DISEASES ASSOCIATED WITH INFLAMMATORY LESIONS OF THE PERIPHERAL ARTERIES*

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BY usage, the term "arteritis" refers to inflammatory conditions of the arterial vessels other than the aorta. Although in recent years significant advances have been made in the knowledge of arterial diseases characterized by inflammatory reactions within and without the vessel walls, this group of disorders still represents a difficult and confusing problem to many physicians. This report discusses only the more important conditions.

To understand the arteritides, consideration of the following facts is essential:

The arteries, like other organs, respond to many pathogenic agents, but with limited types of physiologic and tissue reactions. Several noxious agents of varied character often produce identical types of physiologic and morphologic vascular changes. For example, the same type of acute arteritis can be induced by infections, transplanted homologous adrenal glands, vasospastic drugs and exposure to cold. Hence, from a given type of histologic reaction alone, conclusions about the specificity of the pathogenic factors cannot necessarily be drawn.

The same type of arteritis can be associated with more than one clinical picture. Therefore, the morphologic knowledge of these diseases should not be confused with the clinical aspects of the problem. It is only recently that the clinical features have been adequately studied. In the past, interest has been mainly morphologic.

Acute arteritis is not always of infectious origin, because, as in other tissues, infection is but one of the causes of inflammation. Thus, persistent spasm, like that induced by ergot, causes an acute arteritis that is identical in morphologic composition to that caused by certain virus diseases.

During the healing stage, acute arteritis usually changes into fibrous and degenerative lesions. Again, the characteristics of the healing process are usually the same in a number of diseases affecting the arteries. Arteriosclerosis is therefore merely the chronic tissue response to several different types of persisting noxious stimuli.

Various types of injury to the endothelial layers of the arteries are apt to be followed by thrombosis. Thromboangiitis obliterans is accordingly the result of several types of arterial disease.

Although cellular reactions in the myocardium, — called "Aschoff's bodies," — verrucous endocarditis and thickening of the cardiac valves and of the chordae tendineae occur particularly often in rheumatic fever, these cardiac lesions are not absolutely specific of this disease alone. They occur also in certain other conditions associated with affection of the arteries, skin, serous membranes and joints.

PERIARTERITIS NODOSA

Arteritis associated with perivascular reactions occurs under many conditions. Thus, disseminated lupus erythematosus, dermatomyositis, typhus fever, rheumatic fever, multiple sclerosis and chronic pyelonephritis can be associated at times with localized arterial lesions like those of periarteritis nodosa. Periarteritis nodosa, however, in a *clinical sense*, as originally described by Kussmaul and Maier,[1] refers to a chronic systemic disease of obscure origin and, usually, with a serious prognosis. At present, about 350 to 400 cases of periarteritis nodosa have been reported in the literature. In only 50 or 60 cases, however, has the diagnosis been made clinically. Now that the clinical aspects of the disease are becoming crystallized, this number may be expected to increase rapidly.

From an analysis of the literature, the following general facts emerge. The disease is probably not so rare as observers have thought, and is three to four times commoner in the male than in the female. It can occur at any age, cases having been reported as early as three months and as late as seventy-eight years.[2] It usually occurs between the ages of ten and forty. The disease is not invariably fatal, and it is probable that the prog-

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nosis will improve, when the disease is oftener recognized clinically. The clinical picture varies strikingly, depending on the distribution, severity and persistence of the vascular lesions. Practically any organ can be affected and can be the source of symptoms or signs. It is consequently essential that in patients with bizarre and obscure symptoms suggesting infectious, rheumatic or cachectic disease the possibility of periarteritis nodosa always be considered. The onset is usually gradual. Remissions, long intermissions and exacerbations are common. If fever, cutaneous or subcutaneous nodules, aneurysms of either the large or small arteries, neuritis, asthma, cardiac and abdominal symptoms, pain in muscles and joints, and eosinophilia are present, the clinical diagnosis is more suggestive, but it must be realized that these so-called "characteristic features" are often absent entirely or for a long time. The nervous system is involved more frequently than is usually suspected. Arterial hypertension is present in only a small percentage of the cases, and this depends on an ischemic involvement of the renal arteries. The renal arteries, on the other hand, are frequently involved, although there is no hypertension. The value of recognizing the skin manifestations lies in the fact that such recognition makes diagnosis of the disease possible by biopsy. The veins, as well as the arteries, are occasionally affected.

In 1936, Spiegel[3] reported 17 cases and discussed the clinical aspects of the disease. She emphasized the fact that 7 of the 17 patients suffered from recognized prodromal illness of a "banal" type, such as acute tonsillitis, acute sinusitis and sensitization asthma, which then — without any intervening period of good health — merged with symptoms attributable to periarteritis nodosa. Scarlet fever of atypical form occurred in 2 other patients in the two months preceding the appearance of arteritic symptoms.

In 11 patients, abdominal pain was the first symptom attributable to the vascular lesions. Almost simultaneously, 5 suffered from arthritic pain, and 2 others had cardiac and nephritic symptoms. One suggestive diagnostic association was the combination of abdominal pain and apparently unconnected symptoms such as arthritis, nephritis and cardiac insufficiency. All the 15 cases with post-mortem studies showed cardiac involvement. Twelve had lesions of the coronary arteries of varying degree, and 4 had Aschoff bodies. One patient presented the clinical characteristics of an indeterminate type of verrucous endocarditis. Periarteritis nodosa of the pulmonary arteries was present in 5 cases, and it is of interest that in 6 there were pulmonary lesions not directly dependent on arteritis. Pleural pain, asthma and typical bronchopneumonia may be clinical manifestations. In some of the patients, the roentgenologic findings suggested bronchopneumonia or small pleural effusions. It is of interest that in Spiegel's series 7 patients had surgical complications because of vascular lesions of the gastrointestinal tract. Hemorrhagic pancreatitis occurred in 3 cases. As many as 8 patients had neurologic symptoms; in 6, there was a clinical impression of encephalomeningitis. The lesions indicated productive perivascular and meningeal reactions.

One or more of the following lesions occurred in 12 patients: erythematous macular papules, purpura, urticaria, herpes, edema, pulsatile nodules and white-centered petechiae. Retinal exudates, neuroretinitis and thinning of the retinal arteries occurred in association with renal involvement. One patient showed a characteristic nodule of the choroid on microscopic examination. Twelve patients had polyserositis. The leukocyte count reached 54,000 in 1 patient; polymorphonuclear leukocytes were predominant, although eosinophils and monocytes were also noted.

Leishman[2] has analyzed the clinical features of the cases reported in the literature. The symptoms and signs in order of frequency are as follows: fever (100 to 101°F.), weakness, albuminuria, cylindruria (hyaline and granular), leukocytosis, anemia, tachycardia (out of proportion to the fever), abdominal symptoms, polyneuritis, polyserositis, edema, cutaneous lesions, cardiac signs, enlargement and tenderness of the liver but not the spleen, respiratory symptoms and albuminuric retinitis.

More recently, Grant[4] has made an excellent study of periarteritis nodosa. He points out that the condition is less acute and less frequently fatal than is generally thought. Thus, of 7 patients in whom the disease was clinically diagnosed in 1937, only 3 died in 1940. He studied the characteristics of the vascular lesions and described them as a necrotizing arteritis or a hyaline or fibrinoid necrosis of the arterial wall, with surrounding inflammatory reactions. The necrosis affects mainly the inner part of the media and the subintimal regions, although the whole thickness of the wall may be involved. Either a segment of the arterial wall or its entire circumference may be affected, but commonly only short stretches of the vessels are attacked. The points where the smaller arteries branch off are particularly susceptible. The portions of the artery involved become disorganized, and the internal elastic lamina breaks up, with the result that aneurysms may form. Hemorrhages may occur within the arterial walls or in the sur-

rounding tissues. Thrombi often form within the vessels. The adventitia and surrounding tissues may be swollen or may, on the other hand, show little change. The type and degree of cellular reaction varies considerably, depending on the cause and the stage of the disease. In some cases, the histologic picture is almost that of purulent inflammation; in others, there is but a slight reaction. Usually, the reaction is subacute and contains polymorphonuclear and mononuclear cells, lymphocytes and plasma cells. Giant cells are present in some cases, and eosinophils may occur. In some cases, the arterial lesions may heal, forming granulation and scar tissue, with the result that the wall becomes thickened and the lumen narrowed. No features distinguish this healing process from other types of healing or healed arteritis.

The lesions often develop in crops. Usually, the small arteries and arterioles are affected. The larger arteries become involved indirectly through their nutrient arteries. It is relatively rare for the lesions to be visible to the naked eye. Because often only a small stretch of the artery is involved, it is advisable to examine the excised tissues in serial section, not to miss the affected vascular areas. Rarely, the veins are also affected. The arteries of the heart, kidneys, gastrointestinal canal, pancreas, liver, spleen, skeletal muscles, skin and central nervous tissue are usually the seat of the disease. The facts that necrotic foci of the skin and the myocardium can occur without vascular lesions and that vegetations may rarely develop over the endocardium indicate that tissue lesions can develop independently of vascular lesions. Degeneration, infarcts, necrosis, fibroses and hemorrhages may occur in various organs, depending on the location and specific characteristics of the vascular lesions. Ulcerative lesions of the gastrointestinal canal, nephritis, nephrosclerosis, fibrosis of the liver, nerve degeneration and cutaneous lesions may develop.

According to Grant's experience, the clinical onset of the disease is gradual. Malaise and rheumatic pains, with or without fever, may be the only symptomatic manifestations for a long time. Fever may be absent, low or septic. There may be considerable weakness and wasting. Skin lesions, if present, show much variation: simple erythema, erythema multiforme or nodosum, subcutaneous nodules, and pustular or necrotic lesions. Respiratory manifestations such as bronchitis, asthma, bronchopneumonia, pleurisy and infarcts are relatively often present. Tachycardia, congestive failure, nonbacterial endocarditis, pericarditis and angina are the usual cardiac signs. An unexplained and rather rapidly progressing hypertension has indicated the diagnosis in a few cases. In about 80 per cent of the cases, albumin, hyaline and granular casts, a few red cells, gross hematuria and renal failure occur in various combinations. On rare occasions, Raynaud's syndrome, with or without subsequent gangrene, may occur. Digestive symptoms, such as vague abdominal discomfort, pain, constipation and diarrhea, are frequently present. At times, a so-called "acute abdomen" indicates surgical intervention. There may be blood or mucus in the stools. The ulnar, radial and anterior tibial nerves are particularly affected by the disease.

Ketron and Bernstein[5] studied the cutaneous manifestations of periarteritis nodosa. In their experience, the skin was affected in only about 25 per cent of the cases. The nodules appeared within the skin or subcutaneously, were usually not painful, and could disappear within several days. Erythemas, including scarlatiniform rashes, purpura and vesicular lesions were also present. If the larger cutaneous arteries were involved, the vascular lesion was usually typical, but lesions of the smaller arteries were often not characteristic.

The interrelation between asthma and periarteritis nodosa has been investigated in a selected group of cases by Rackemann and Green.[6] Of the 8 cases reported by them, all were women but 1. This is of interest because in the unselected groups the males predominate. Six patients developed asthma before the age of thirty-six years, and in most the course was short. In all the cases, the asthma was intrinsic in type. Four of the 8 patients had had trouble with the nose and sinuses. It is of particular interest that each of the 8 cases with asthma had an intense type of eosinophilia, in 3 reaching as high as 70 per cent of the white blood cells. The total white-cell count was also elevated, sometimes to 25,000 and in 1 case to 50,000. It should be pointed out, however, that this group was selected according to the observers' special interest in asthma. A study of the literature indicates that asthma does occur, but only in 10 per cent of the cases, and even when present is not always associated with eosinophilia.

Kernohan and Woltman[7] have studied the nervous system in 5 cases with periarteritis nodosa. In 3 cases, the peripheral nervous system was widely degenerated; in 1, the brain, and in 1, the choroid of the eye was involved. In only 1 case were the nutrient arteries to the peripheral nerves affected in the inflammatory process. The degeneration of the peripheral nerves appeared to be the result of occlusion or marked narrowing of the lumina of the nutrient arteries to the nerves. The degeneration, which was diffuse at lower levels, began as infarction at higher levels. No inflammation of the nerves was observed.

More recently, involvement of the central nervous system in periarteritis nodosa has been discussed in the German literature, but some of these publications have not been available. Stanojevic and Dimitrijevic[8] reported a case with involvement of the central as well as the peripheral nervous system. There were also changes in the eyegrounds designated as "neuroretinitis angiospastica," with early manifestations of retinal detachment. Since the patient also had hypertension, it is questionable whether these eyeground changes were specific. Judging from a study of the literature, I believe that these changes, if they do occur in periarteritis nodosa, usually depend on the associated hypertension. Rarely, however, as in the case reported with histologic findings by Gjertz, Nordlöw and Svenmar,[9] the affection of the retina depended on typical lesions of the vessels. It is of interest that this patient also suffered from episcleritis.

Sawyer,[10] Allen[11] and Mondor, Ducroquet and Olivier[12] have reported on the surgical aspect of periarteritis nodosa. Occlusion or aneurysmal rupture of large or small arteries of various abdominal organs may lead to symptoms and signs imitating those of penetrating peptic ulcers, chronic cholecystitis, perinephritic abscess, pancreatitis, mesenteric infarction and peritonitis. Even the liver may be affected.

Thromboangiitis Obliterans

Although this arteritis was described as "endarteritis obliterans" by von Winiwarter as early as 1879, and although it has been intensely studied since 1908 by Buerger and others, several significant aspects of the disease remain obscure. Not only is its etiology unknown, but the chronic stage of the disease is still confused with certain types of arteriosclerotic degeneration of arteries of the lower extremities. One needs particularly to know more of the early stage of the disease. It is surprising how few studies of biopsied specimens are available from this stage, when not only are the symptoms vague (paresthesias) but laboratory methods may even fail to demonstrate ischemia. In my experience, during the acute and the early stages, several characteristics of the disease may closely simulate those of periarteritis nodosa. At times, only the physiologic evidence of involvement of the larger arteries of the extremities may be the differential point.

In the recent literature, controversy about the etiology of thromboangiitis obliterans continues. It is probable that it can be caused by a number of factors, and frequently several of these factors seem to act synergistically. Recently, Goodman[13] brought forward further evidence in favor of the rickettsial origin of the disease in at least one group. He claimed that thromboangiitis obliterans is a late manifestation of typhus fever. Gangrene as a direct complication of typhus fever has been reported repeatedly in the literature. Goodman also claimed that wherever typhus fever is endemic, there is a high incidence of thromboangiitis obliterans. In 100 cases of thromboangiitis obliterans studied by him, all patients gave a positive skin reaction following the intracutaneous injection of the rickettsia suspension. In 93 per cent of a corresponding number of control cases, this test was negative. In 100 cases of Brill's disease, it was 97 per cent positive. It is of interest in this connection that, in 1939, Parker[14] reported thromboangiitis obliterans as one of the complications of Rocky Mountain spotted fever and mentioned the frequency with which gangrene of the extremities was observed in animals inoculated with the rickettsia of this disease. Jordanus, as quoted by Goodman, described the sixteenth century epidemic of typhus that swept the Continent from Russia and caused millions of deaths. He mentioned as its chief features high fever, prostration and gangrene. Goodman also stated that in Japan "juvenile gangrene" is very common, and that rickettsial diseases, particularly tsutsugamushi, are likewise common. The vascular lesions in typhus fever, as described originally by Fraenkel,[15] consist of necrosis of the media of the small arteries within the cutaneous roseola, often associated with cellular reactions around the vessels. Fraenkel states that these vascular reactions remind one of those found in periarteritis nodosa, although the two diseases are not related to one another.

Arterial thrombosis of the extremities can occur, however, in a number of contagious diseases. Hoyne and Smollar[16] report the occurrence of gangrene of the lower extremities as a rare complication of scarlet fever. These writers were able to find in the literature 17 cases of such gangrene associated with scarlet fever. In 5 of these, the gangrene was bilateral, and in 16 it was so-called "dry gangrene." In 1 case reported by Hoyne and Smollar, there was an acute myositis above the area of gangrene, associated with panarteritis of most of the arterioles. In another, reported by Hoyne in 1915, dry gangrene of all four extremities developed, and in addition both ears and both cheeks were affected. It has been estimated that gangrene of the extremities occurs only once or twice in 30,000 cases of scarlet fever. This complication does not depend on the severity of the illness, but is considered as an unusual arterial response of the host.

In recent years, an increasing number of cases of thromboangiitis obliterans with central-nervous-system manifestations have been reported. These nervous symptoms may appear during either the acute or the chronic (arteriosclerotic) stage. Lindenberg[17] reports the affection of the central nervous system in this disease. He states that thromboangiitis obliterans is primarily a morphologic condition due to several agents. The effect on the central nervous system manifests itself in granular atrophy, with multiple small softened areas. In some cases, the distal small branches of the larger cerebral arteries are affected, whereas in others the vascular affection shows completely irregular involvement. Histologically, the lesions represent an endarteritis with thrombi. The media is relatively free. A perivascular inflammatory reaction may also be present. In chronic cases, the intima of the involved arteries is thickened. Recanalization of thrombosed arteries occurs frequently. The clinical manifestations are varied. Transient headaches, palsies, syncope and convulsions are prone to occur. The disease may imitate Alzheimer's disease.

The interrelation between thromboangiitis obliterans and the retinal vessels is of some interest. This topic has been discussed and reviewed recently by Wagener.[18] According to Wagener's report, Marchesani was impressed by the association, in a group of young persons, of recurrent hemorrhages into the retina and vitreus on the one hand, and transitory severe cyanosis of the extremities and trophic disturbances in the nails and skin on the other. In a number of these patients, there were no pulsations in one or more of the arteries of the lower extremities. All showed dilatation of the venous loops of the capillaries of the nailfolds, often associated with capillary hemorrhages. In the series of 22 patients reported by Marchesani, 2 suffered from gangrene of the toes, and 1 from gangrene of the fingers; 19 had evidence of "latent" Buerger's disease. In 1 case with gangrene, histologic examination revealed variable narrowing of the lumens of the smallest retinal vessels, which was due to swelling of the subendothelial tissues. In some of the larger arteries and veins, the endothelial layers were destroyed and the lumens filled with fibrinlike material. Other lumens contained material rich in cells. It is interesting that vessels of the iris and of the ciliary body were also involved. Marchesani suggested that these lesions of the vessels of the eyes were the ocular manifestations of Buerger's disease. Three of Marchesani's patients with primary symptoms of retinal periphlebitis showed atypical multiple sclerosis. Wagener states that in the Mayo Clinic in a large series of patients with Buerger's disease he had but few cases of thrombosis of the retinal vessels. In 1 patient with presenting features of recurrent hemorrhages into the vitreus, typical manifestations of Buerger's disease developed years later. Other reports in the literature on the interrelation of eye symptoms associated with recurrent hemorrhages in the vitreus or vascular lesions in the retina on the one hand, and vascular affection of the extremities on the other, are summarized by Wagener. It is questionable whether in all these cases the ocular disturbances are manifestations of thromboangiitis obliterans, as originally suggested by Marchesani.

Since the chronic manifestations of thromboangiitis obliterans are adequately recognized, and since no outstanding contribution has been made recently to the knowledge of this phase of the disease, discussion of it will be omitted.

Temporal Arteritis

Since the first case of temporal arteritis, reported in 1931, further cases of a similar nature continue to appear in the literature. Recently, Sprague and MacKenzie[19] have reported the case of a sixty-six-year-old man who complained of temporal neuralgia after a vague onset, and subsequently of temporomandibular arthralgia. The fact that in this patient, as has been true in other cases, the localized arteritis was associated with generalized debilitating conditions indicates that arteritis is a manifestation of a generalized disease. Bowers[20] summarizes the histologic findings in temporal arteritis. That the medial coat is usually affected is indicated by the presence of necrosis, at times with hemorrhage. The destroyed areas are replaced by granulated tissue in which giant cells are present. If all the coats are involved, there is intimal thickening and round-cell infiltration of the adventitia. The process may extend to tissues around the arteries. The lumens of the vessels may become closed. Both sexes are affected, and the patients are usually elderly, with signs of hypertension and arteriosclerosis. The cause of the disease is unknown, and its duration may range from five to six months, with some tendency to relapse, but with eventual complete recovery — in my experience, the duration has at times been but a few weeks. The cerebral, retinal and radial arteries can also be involved. In the case reported by Bowers, the occipital arteries were also affected.

Symptoms such as dull throbbing headaches, which become worse at night, fever, night sweats, poor appetite, loss of weight, anemia, weakness and mild leukocytosis may precede by several weeks

local signs of inflammation of the arteries. Small nodules may be felt over either the main branches or the smaller ones.

Recently, Hoyt, Perera and Kauvar[21] reported 3 cases of acute temporal arteritis from the Peter Bent Brigham Hospital. This brings the number of cases reported in the literature to over 20. They point out that in temporal arteritis the presenting symptom is usually a severe, boring headache referable to both sides of the head, often severer on one side than on the other. Often, too, the pain is more intense at night, and frequently neither morphine nor barbiturates succeed in abolishing the pain. The temporal regions and temporal arteries may be tender, and during the course of the illness the vessels may become enlarged, tortuous and surrounded by areas of hyperemia. Tenderness and painful mastication were prominent features in one of the cases reported. The severity of the illness is often entirely out of proportion to the degree of local disease present. These authors suggest that the inflammation of the temporal artery is perhaps but a local manifestation of a more widespread disease. The histologic findings on biopsied arteries in these cases were the same as those described above. It is interesting that from a practical standpoint the most effective treatment seems to be removal of a segment of the temporal artery. After such therapy, the headache is relieved, even though the fever and generalized symptoms may continue for weeks. This local benefit from biopsy probably depends on section of the sympathetic nerves.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTE-MORTEM AND POST-MORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor*

CASE 27411

PRESENTATION OF CASE

A thirty-year-old single American woman was admitted to a mental hospital, where she remained until her death twenty years later.

The patient's family and personal histories were irrelevant. Her birth, infancy and early childhood were normal. She entered school at six and was considered very bright, but began to show a tendency toward seclusiveness and did not make friends easily. She enjoyed quiet, solitary diversions, was extremely sensitive, did not care for the company of men, and never had any love affairs. At the age of nineteen, the patient showed a definite change in her behavior: she cried a great deal, and was unable to give any reason for the change. At times she was hysterical, and could not control herself. This condition became progressively worse, and she began to lose interest in things she had previously liked. Finally, she would not talk at all. She then began to smile and laugh to herself, mumbled and spoke incoherently in low tones, and for several years would not converse. At the age of twenty-one, a panhysterectomy was performed on the recommendation of a psychiatrist. There was no further change in her general condition except that she gained about 100 pounds in weight. It was stated that until approximately one year before admission she was impulsive, violent and destructive. From the time of her institutionalization, when her physical and neurologic examinations were negative, until her death twenty years later, she showed progressive deterioration and reduction of activities. Eight years before death, the patient acted as if she had pain and tenderness in the gall-bladder region, and there were clay-colored stools. These symptoms persisted for a few weeks, then passed uneventfully. Two years later, the patient was placed on a diet and lost 85 pounds, the eventual weight being 165 pounds. Her condition was unchanged, and her general health appeared excellent. She was entirely out of communication with her physicians, mumbled to herself, fidgeted, and played with trinkets and toys. A Graham test showed no visualization of the gall bladder, but the examination was not considered entirely satisfactory because of the large size of the patient and her general lack of co-operation. At this time, roentgenographic studies showed kidney outlines well visualized on both sides and normal in size, shape and position. Low in the pelvis on the left side, there were four small rounded areas of calcification, which had the appearance of phleboliths. Overlying the upper pole of the left kidney there was a group of irregular dense shadows, which could have represented renal calculi or calcified mesenteric lymph nodes. Repeat studies one year later again showed these several areas of density in the soft tissues, which were thought probably to represent calcified mesenteric nodes. No masses were felt on physical examination. The patient's condition remained approximately unchanged.

Eighteen months before death, the patient had, for the first time, an epileptiform convulsion accompanied by incontinence of urine and feces. The convulsion was generalized, and following it the patient seemed confused and drowsy. There were no definite neurologic findings, and no apparent headache, papilledema, stiff neck or localizing signs.

An electroencephalogram was suggestive of a pathologic process in the left occipitoparietal region. X-ray films of the skull were unusual but not definitely informative. There was irregular thickening of the inner table of the frontal bone, owing to hyperostosis, with unusually extensive calcification of the falx, but without evidence of chronic intracranial pressure.

Fifteen months before death, during the course of a routine physical examination done at the mental institution, an abdominal mass about 5 cm. in diameter was noted in the right lower quadrant. It was considered to coincide with the position of the calcified areas of density observed by x-ray study, and no further diagnostic procedures were attempted. On rectal examination, an extrinsic mass was felt about 5 cm. above the anus. Subsequent examinations showed several masses, about the same size, all palpable, and apparently increasing in size. Repeat stool examinations were negative for occult blood, the last test being done nine months before death.

The patient's condition continued with very little change until about six months before death, when she began to seem weak, and appeared to favor her abdomen as though there were pain or tenderness there. She vomited several times. There was no fever, and no constipation. The abdominal masses seemed somewhat larger. She failed gradually, showing physical deterioration. One month before death, she had "difficulty" with

respiration and developed a temperature of 102 to 104°F. Examination of the chest revealed dullness to flatness at the right base, with diminished breath sounds, and a roentgenogram taken with a portable machine showed evidence of fluid in the right pleural cavities and a linear band of density in the lung field, extending down from the right hilus.

The temperature from this time continued to fluctuate between 98.6 and 102.0°F., and physical findings in the chest increased; the patient became weaker and developed mild dyspnea. She remained about the same for three weeks, and then quickly failed and died.

FIGURE 1. *Intravenous Pyelogram Showing Calcified Masses Superimposed on the Right Kidney Shadow.*

DIFFERENTIAL DIAGNOSIS

DR. JOE V. MEIGS: It would be presumptuous for a surgeon to attempt to discuss what kind of insanity this patient suffered from. It is evident even to the layman, however, that it was a major psychosis that demanded institutionalization. I cannot remember ever to have heard of attempts to cure insanity by such an operation as this. It is very unfortunate that we do not know the details of the operation, since the term "panhysterectomy" has been applied to different operations at different times. As used today, it means that the uterus and tubes were removed and the ovaries were left behind. In 1910, I should suppose that it meant that the ovaries had also been removed. I think we can assume that the menses ceased, since we are given no information about them. The insanity naturally made it impossible to inquire in detail about subjective symptoms, such as periodic sensations in the breast and hot flashes, which would allow us to guess whether or not an artificial menopause was produced. It is obviously of great importance to know whether or not the ovaries were removed. I shall guess that they were, and am basing this assumption chiefly on the fact that the patient gained 100 pounds in a brief period after the operation. Of course, one can gain a great deal of weight after

any operation. One feels better and eats better, and the operation is an excuse for not exercising. However, a weight gain of 100 pounds is a striking phenomenon and definitely suggests that endocrine function was disturbed.

The gall-bladder symptoms are reported to have persisted only a few weeks; they then passed uneventfully. This seems to me important because we are looking for all possible sites of primary neoplasm, to explain the subsequent history. But since the symptoms lasted only a few weeks, since we know that an unsatisfactory Graham test was later performed, and since the patient lived six years after the attack, I believe that there was nothing of serious consequence in the right upper quadrant. A report of an x-ray examination at this time suggested calcified mesenteric lymph nodes on the left side of the pelvis.

Dr. James R. Lingley: This is the plain abdominal film taken six years before death. Low in the pelvis on the left, some small round areas of calcification, which at the time we thought were phleboliths, are visible. Higher up on the same side is a larger group of dense shadows that overlie the upper pole of the left kidney; these could be but probably are not renal calculi. They could be calcified mesenteric nodes. These films were taken six years later, and between the two examinations a remarkable change in the appearance of the abdomen had occurred. Unusually large areas of calcification were scattered throughout the abdomen. Here is one in the mid-pelvis, which appears to form the upper border of a soft-tissue mass. Another in the right upper quadrant has somewhat the appearance of a staghorn calculus. It lies outside the kidney outline, however, and above it (Fig. 1). Here in the mid-line is a large mass, which runs parallel to the transverse colon but was shown by a barium enema to be definitely outside the bowel. It was fairly movable, and we assumed that it was in the mesentery. In all these areas, the type of calcification is very unusual. It does not have the appearance of solid calcium but looks more as if the calcium were deposited in innumerable small particles throughout the soft-tissue masses.

Dr. Meigs: That was a great change — much greater than I had realized on reading the history. I note that they speak of a mass 5 cm. in diameter, which was felt in the pelvis. That is only about the size of a golf ball, and if they could feel it in a patient who weighed 165 pounds it was probably even larger. The statement that repeated stool examinations were negative for blood is very important, and permits us to rule out a primary tumor of the bowel. Evidently in the last few months the masses in the abdomen grew rapidly, and the patient began to go downhill, finally developing what might have been pneumonia, fluid in the chest or empyema. We are at a great disadvantage in not knowing certainly whether any ovarian tissue was left. If she still had her ovaries, one might think of a tumor primary in the upper abdomen, starting at about the time that she had the right upper-quadrant difficulty, that metastasized to the ovaries and filled them with tumor analogous to a Krukenberg tumor. One also thinks of the possibility of a primary ovarian tumor, with metastases throughout the abdomen, including the omentum; this would account for the movable mid-line tumor. Malignant ovarian tumors, particularly papillary adenocarcinomas, not infrequently undergo extensive calcification. Certainly that possibility cannot be ruled out. I have already committed myself, however, to the assumption that both the ovaries were taken out at the original operation. I do not know of any other neoplasms that calcify as extensively as this, and I must therefore fall back on the diagnosis of tuberculosis. I do not recall ever having seen a case of abdominal tuberculosis that progressed like this, but it remains my best guess and on that basis I assume that the final episode in the chest was probably a tuberculous pleurisy.

Dr. Tracy B. Mallory: What would you say about the probability of tuberculosis, Dr. Holmes?

Dr. George W. Holmes: I am familiar with this case and consequently cannot give an unbiased opinion. I should like to point out that these are very unusual shadows. I cannot remember ever to have seen anything like this picture. Certainly it is not typical of tuberculosis.

Dr. Wyman Richardson: I saw this patient two or three times and made a diagnosis of "rocks" in the abdomen of unknown etiology. The clinician who saw her placed most emphasis on the possibility of tuberculous adenitis, but I think almost everyone was very much confused about the diagnosis.

Clinical Diagnoses

Schizophrenia.
Malignant disease, undetermined?
Tuberculosis of retroperitoneal lymph nodes?

Dr. Meigs's Diagnoses

Tuberculosis of the abdominal lymph nodes.
Tuberculous pleurisy.

Anatomical Diagnoses

Papillary cystadenocarcinoma of the ovary, with carcinosis of peritoneum and pleura and metastases to serous coats of intestines, liver, spleen and gall bladder.
Cardiac infarction.
Mural thrombus, right ventricle.
Pulmonary thrombus, left lower lobe.
Pulmonary infarct, left lower lobe.
Pulmonary atelectasis, bilateral, marked.
Hydrothorax, bilateral, marked.
(Schizophrenia.)
(Epilepsy.)
Frontal exostosis.
Ossification of falx cerebri.
Cholelithiasis.
Cortical cysts of kidney.
Hydronephrosis, left.
Hydroureter, left.
Operative scar: hysterectomy.

Pathological Discussion

Dr. Mallory: The autopsy proved one thing definitely — that it was not tuberculosis but metastatic tumor. All these tumor masses were extensively calcified. The calcification was mostly of a fine gritty character; gross examination suggested the type of calcification that is not infrequently seen in certain ovarian tumors and is due to very minute spherical masses of calcium known as psammoma bodies. When the microscopic sections came through, it was evident that the tumor was a papillary adenocarcinoma exactly similar in appearance to those that arise in the ovaries, with a great many psammoma bodies in it. It had metastasized very widely. We found many nodules scattered over the mesentery and on the bowels; apparently, these had started on the serosa and worked inward, some actually reaching the mucosa and almost obstructing the lumen. There were pleural metastases, which probably accounted for the chest fluid. The pelvis was totally filled with tumor material, and multiple sections at very close intervals failed to show anything that could be recognized as uterus, tubes or ovaries. If it were not for the story of the "panhysterectomy," I should assume that this was an ovarian tumor. We found no other probable primary site, and I have not happened to see a tumor of this type arising in any other tissue. Apparently, death was not due to the tumor but to a combination of a large infarct of the heart and pulmonary embolism. The skull showed extensive hyperostosis of the frontal bone, and the brain was grossly normal.

Dr. Meigs: I have seen ovarian tumors with calcification visible on x-ray films but never with as diffuse calcification as in these films. I should expect to see small granular areas more widely scattered.

Dr. Lingley: We have seen another case with areas as close as these.

CASE 27412

Presentation of Case

A sixty-two-year-old American schoolteacher was admitted complaining of marked breathlessness.

The patient had frequent sore throats until fourteen years before entry, at which time a tonsillectomy was performed. Subsequently, she had no recurrences. For about twenty-six years she had been slightly breathless on ordinary exertion, although this had not prevented her from carrying on her usual activities. For about five or ten years she had been aware of an irregularity of her heart, which had not been troublesome, however.

The irregularity had been constant so far as she knew. Four years before entry, she was seen by a physician because of bronchitis and was told that she had heart trouble and that her systolic blood pressure was 180. She was advised to take Digitora, one grain twice daily, which she continued to do until the time of entry. In spite of the treatment, the dyspnea had become more pronounced. Six months before admission, the patient consulted a physician for recurrent attacks of "intestinal flu," which were characterized by lower abdominal pain and nausea. The attacks were sufficiently severe to require rest in bed for a few days and sedatives for relief. They recurred at intervals for a few days to several weeks, but for the previous few months they had become less frequent and less severe. There was no associated vomiting, diarrhea or melena. Since the onset of this symptom, dyspnea had become more marked, and the ankles became swollen toward evening. For about four weeks, there were nocturnal attacks of dyspnea, associated with wheezing, which caused her to sit up in bed for relief. Occasionally, she was compelled to sleep in a sitting position. There was slight cough but no bloody sputum, and concomitant with the orthopnea there were a sensation of distress over the anterior chest and a tight feeling across the upper abdomen. For four days, there was marked swelling of the feet and legs up to the mid-thigh.

There had been no previous attacks of rheumatic fever, chorea or related diseases.

Physical examination showed an elderly woman

propped up in bed, with slight cyanosis of the lips, face, fingers and hands. There was distention of the neck veins up to the angles of the jaw. A slight wheezing sound with respiration and occasional attacks of a Cheyne–Stokes type of breathing were noted. The apex impulse was diffuse and heaving, its maximal point being in the fourth interspace, 13 cm. to the left of the mid-sternal line. No thrills were palpable. The left border of dullness was 14 cm. from the mid-sternal line in the fourth interspace, 18 cm. in the third; the right border of dullness was 5 cm. in the fourth interspace. The rhythm was totally irregular, with an apical rate of 100. The pulse at the wrist was 60. The aortic second sound was moderately diminished, and the pulmonic was louder than the aortic and moderately accentuated. The first sound at the apex was marked by a moderate to loud, rather high-pitched systolic murmur, which was widely transmitted. At the apex, there was a short rumbling diastolic murmur immediately following the second sound. The blood pressure was 195 systolic, 110 diastolic. The radial arteries were soft, and the pulse moderately small in volume. Over the right lung base posteriorly, there were dullness and diminished to absent tactile fremitus. The breath sounds were diminished in intensity, but the voice sounds were of good quality. Throughout both lung bases, there were a moderate number of medium crepitant rales. The abdomen was soft, and the liver edge, which extended to the level of the umbilicus, was slightly tender. The spleen was not felt, and there was no evidence of ascites. There was edema of the lower extremities up to the mid-thigh.

The temperature was 100°F., and the respirations were 28.

Examination of the urine showed a specific gravity that varied between 1.008 and 1.014, with a slight trace of albumin. The sediment contained a rare red blood cell, 25 to 50 white blood cells and occasional granular casts per high-power field. The blood showed a red-cell count of 4,900,000 with a hemoglobin of 80 per cent, and a white-cell count of 10,000 with 73 per cent polymorphonuclears. A stool examination was negative. The blood Hinton reaction was negative. An electrocardiogram showed auricular fibrillation, with a tendency to right-axis deviation. There was low voltage and slurring of the QRS waves. T_1 was low, almost flat, and T_2 and T_3 low, with sagging ST intervals. Q_4 was 2 mm., and T_4 low and diphasic.

Three days after entry, shortly after a light lunch, the patient suddenly developed abdominal discomfort and pain. This was relieved by a medicated enema, but shortly after its return, the patient became nauseated and vomited and her skin became ashen. She complained of epigastric discomfort and perspired freely. The blood pressure at that time was 200 systolic, 100 diastolic. There was an increase in moist rales in the chest, and the sputum was faintly tinged with bright-red blood. About fifteen minutes later, this attack subsided, but the patient continued to bring up streaked sputum for the next few days. Despite the administration of digitalis and sedatives, the patient's general condition remained unchanged, and the heart rate was not lessened. There was dyspnea at rest, and the cyanosis and engorgement of the neck veins continued. On the tenth hospital day, a right thoracentesis was performed, and 1360 cc. of straw-colored fluid withdrawn. This clotted before the procedure was completed. Immediately after the tap, the patient stated that a constricting sensation, from which she had previously suffered, had subsided. Subsequently mercurial diuretics were administered, and the patient became more comfortable, although additional chest taps were necessary.

One month after entry, it was noted that nocturnal dyspnea was increasing in frequency and severity. Throughout this time, the temperature fluctuated between 98 and 101°F. Increasing dosage of digitalis failed to control the ventricular rate completely, and a pulse deficit of 40 to 80 continued. Blood-stained sputum was frequently noted. About ten weeks after entry, the patient was suddenly seized with a shaking chill and became markedly cyanotic, and the pulse at the wrist was imperceptible. She was more comfortable shortly afterward, although the temperature rose to 102°F. Three days later, note was made of pain across both sides of the upper abdomen, radiating to the back. The apical heart rate rose to 120, and there was an icteric tint to the scleras. At this time, the white-cell count was 30,000, with 89 per cent polymorphonuclears. The icteric index was 70. The nonprotein nitrogen of the blood was 38 mg. and the serum protein 4.4 gm. per 100 cc. Her condition remained essentially unchanged for the succeeding two weeks, and at that time copious spitting of blood began. This was associated with a slight cough, and the blood apparently welled up in the throat. The expectorated material was almost pure blood, with very little mucus. There was no dyspnea or jugular engorgement, but numerous moist rales were audible throughout both chests. Subsequently, her condition became progressively worse, and she died three months after entry.

Differential Diagnosis

Dr. Edward F. Bland: Here is a woman who had been mildly incapacitated by shortness of breath, presumably referable to her heart, for twenty-six years, from the age of thirty-six to sixty-two, and with irregularity of the heart for the last ten years of her life. We know that it was fibrillation ultimately, and the terminal events consisted in congestive failure and certain complications thereof. I know of but one type of heart disease that is apt to permit such a long life with moderate disability for so many years — rheumatic heart disease. Irrespective of the physical signs, I think we can be reasonably certain of this on the basis of the history alone. From the physical signs described, the patient undoubtedly had extensive involvement of the mitral valve, with regurgitation and stenosis. There was, of course, marked enlargement of the heart. Most of the other signs described are consistent with rheumatic heart disease and heart failure. I suppose that an x-ray film was taken. She could hardly have been in the hospital for three months, with all these things happening, without a chest film. I should be interested in that.

Dr. Benjamin Castleman: No x-ray films were taken.

Dr. Bland: Perhaps the patient was too sick. It would have been of interest to search for calcification of the mitral valve. There probably was some, but it might have shown up only under the fluoroscope, or perhaps not even there. It would have been of further interest to know if we were dealing with something other than cardiac impairment as a contributing cause of the long-standing dyspnea.

The electrocardiogram is of some interest. It shows a little right-axis deviation; I should have expected more. Of course, this patient also had hypertension, which imposed an added strain on her heart, principally on the left ventricle, and hence would tend to lessen the degree of right-axis deviation that she might otherwise have had. The description in the record covers satisfactorily the other electrocardiograms shown here on the screen. There was auricular fibrillation, rather low voltage and very slight right-axis deviation, the degree of which, as noted above, is a little disturbing. There was also a digitalis effect on the T waves, although, as we shall note shortly, the ventricular rate was controlled only with a good deal of trouble.

I should like to speculate a little about the probability of slight scarring of the aortic valve in the presence of such extensive rheumatic involvement of the heart. Nothing in the physical signs described permits one to guess that such scarring occurred, but I think that there is perhaps a fifty-fifty chance of finding a relatively slight degree of aortic-valve involvement.

Certain features of the final illness require some special consideration, and I think there may be some reasonable difference of opinion concerning them. The attacks, called abdominal, before she came to the hospital interest me. They coincided with the beginning of frank congestive failure and consisted in pain and nausea, without other gastrointestinal symptoms. We know that pain in the abdomen with congestive failure is common and most frequently due to demonstrable engorgement of the liver. Sometimes, people have pain from a sore liver oftener at night than during the day, probably because of the semirecumbent position assumed in bed, whereas when they sit in a chair the liver is not quite so encroached on. For a number of years, we have been searching without success for an explanation of the abdominal pain when congestive failure is not evident in patients with well-marked mitral stenosis. Here we need, I think, look no farther than to the congestion of the liver and of the gastrointestinal tract as a satisfactory explanation. Mesenteric embolism seems unlikely.

We now come to a second point, which I think needs a little discussion. These recurring episodes of pulmonary distress, which were associated with an increase in the signs of congestion and one of which was accompanied by an actual chill, together with the vague pain in the chest followed by fever, hemoptysis and icterus, were certainly due to multiple pulmonary infarction. This patient had two very good reasons for having both respiratory distress and hemoptysis. She had obvious congestion of the lungs, and she might have had a little blood in the sputum simply from this. However, these recurring episodes, especially the one followed by icterus, indicate multiple pulmonary infarcts. The degree of icterus in this patient is interesting, for an icteric index of 70 represents considerable jaundice and hence extensive infarction, I should guess. Someone asked me recently if we saw icterus as a result of infarction of organs other than the lungs. At the time, I did not recall such a case.

Dr. Castleman: We have had one case of jaundice associated with infarction of the heart without pulmonary infarction. It was reported several years ago.[1]

Dr. Paul D. White: Yes; I remember that case. I suppose that such a case is related to the extent of hemolysis and the passive congestion in the liver.

DR. CASTLEMAN: Yes; both factors must be present.

DR. BLAND: The next point I should like to comment on is this patient's fever. There are several adequate reasons why she may have had fever. Extensive congestive failure may give rise to a degree or two. We know that the pulmonary infarcts could have caused fever, but the question is raised whether any other infection was present, bacterial or otherwise. Perhaps not, but I shall keep that possibility in mind for a final word later.

Toward the end of her life, the patient had a low serum protein, 4.4 gm. per 100 cc., which is within the edema range. She had several obvious causes for a low serum protein. Undoubtedly, the dietary intake for one reason or another had been inadequate. She had congestion of the liver and had lost fluid by both diuresis and tapping. All these factors might have contributed. Then we come to the question of some obscure infection in the heart as a possible added factor in her demise. In a young person, heart failure complicating a rheumatic heart is a fairly reliable sign of underlying infection of some sort or other, but this patient was too old to apply this general principle without other supporting evidence, so that we have to discount the presence of congestive failure as a possible indication of active infection, bacterial or rheumatic. A good deal of difficulty was obviously encountered in controlling the ventricular rate. Among the things in general that make it difficult to control the ventricular rate when fibrillation is present are infection, infarction in the lung and thyrotoxicosis. There is no reason to suspect thyrotoxicosis. I think that the patient had multiple infarction of the lungs, which is probably the best explanation.

A final word about the lungs: I was puzzled why the patient bled so much from her lungs at the end, since there was not much evidence at that time of gross congestion and since, in fact, the pulmonary congestion seemed temporarily better; yet she raised almost pure blood, without sputum and with very little cough. That is a little unusual, but perhaps the pulmonary circulation was better because she was raising blood. We know that patients with rheumatic heart disease and mitral stenosis are prone to bleed from the lungs from either infarction or congestion and at times without clinical evidence of either. I think it unlikely that Dr. Castleman will tell us later that the patient had a rheumatic pneumonitis. Although I do not like to discard it without some further comment, I do not believe I could make a diagnosis of acute rheumatic fever.

Did she have bacterial endocarditis? I do not believe so for several reasons. She had auricular fibrillation. That does not exclude endocarditis, but the combination is a little unusual. She had chronic congestive failure for at least nine months. That would be most unusual if the heart failure were due to bacterial infection. Did she have some chronic disease of the lung to explain her respiratory symptoms rather than chronic heart disease? Probably not. She had respiratory symptoms for too many years, with no clubbing of the fingers and no suggestive sputum during that time.

In summary, I should say that in spite of the patient's age she had chronic rheumatic heart disease, with extensive mitral involvement, both stenosis and regurgitation. She had a complicating hypertension, which may have been an added factor in the later downhill course and ultimate death, but the rheumatic disease of the heart was, I believe, of first importance. She died of chronic congestive heart failure, with recurring pulmonary emboli and infarction.

DR. HOWARD B. SPRAGUE: Abdominal pain in these patients with cardiac failure bothers me as much as it does Dr. Bland. Some of them who do not have much enlargement of the liver may have acute engorgement causing pain. Also, we have been surprised to find renal infarcts in these patients. I want to ask Dr. Castleman, Is it possible to get small enough emboli to the blood supply of the intestines to have attacks of pain without infarction of the intestinal wall?

DR. CASTLEMAN: I do not believe so. Infarction is found only when the larger vessels are occluded, because ordinarily there is enough collateral circulation between the intestinal branches to prevent it. I suppose emboli in the mesenteric branches are not uncommon, but I do not believe they have been hunted for seriously enough.

DR. WHITE: With reference to renal infarction, Dr. Reno R. Porter and I[2] found that renal infarcts are usually painless. The majority of patients whom I have seen with abdominal pain complicating heart failure have had distention of the intestine from gas due, in part at least, to stasis. They usually have liver engorgement also, but not always. Of course there is something here besides gas.

CLINICAL DIAGNOSES

Valvular disease, chronic, cardiac.
Mitral stenosis.
Hypertension.
Pulmonary infarction.

Dr. Bland's Diagnoses

Rheumatic heart disease.
Mitral stenosis and regurgitation.
Auricular fibrillation.
Congestive heart failure.
Multiple pulmonary infarcts.
Hypertension.

Anatomical Diagnoses

Rheumatic heart disease, chronic, with mitral stenosis.
Cardiac hypertrophy.
Mural thrombus, left auricle.
Thrombosis of popliteal vein, left.
Pulmonary embolism, bilateral.
Pulmonary infarction, right lower lobe.
Icterus.
Renal infarction, multiple, bilateral, healed.
Arteriosclerosis, marked aortic, moderate coronary.
Nephritis, chronic vascular, marked.
Chronic passive congestion of liver and spleen.
Pulmonary congestion.
Hydrothorax, bilateral.
Peripheral edema.
Peritonitis, chronic fibrous.
Cholelithiasis.
Dilatation of bile ducts.
Focal necrosis and abscess formation of pancreas.

Pathological Discussion

Dr. Castleman: This patient had a large heart, weighing 800 gm., with marked dilatation and hypertrophy of the right auricle and ventricle. The right ventricle measured 5 mm. in thickness, which I think would be enough to cause right-axis deviation.

Dr. Bland: It is not entirely a question of the thickness. It is a question of disproportion between the normal ratios of the two ventricles.

Dr. Castleman: The left measured 17 mm. The mitral valve was markedly stenotic and calcified, although the opening measured 3 cm. in circumference. In the left auricular appendage was a mural thrombus, and there were small healed renal infarcts that may have had something to do with the pain, but I doubt it. There were several large infarcts in the lower lobe of the right lung involving about two thirds of the lobe; the other lobes were normal. The extent of the infarction, together with the congestion of the liver, certainly accounts for the icterus. There was about 200 cc. of fluid in the right pleural cavity, and 50 cc. in the left. In addition, old adhesions were found all over the abdomen, especially in the region of the gall bladder, which was filled with two large stones and inspissated bile. The pancreas showed several areas of acute fat necrosis, and I believe the abdominal pain was due to some disturbance in the biliary tract associated with chronic pancreatitis. Small foci of old fat necrosis are occasionally seen without symptoms, but here it was much more acute than usual.

Dr. White: Was there any active myocardial rheumatic disease?

Dr. Castleman: No; we were unable to find any evidence of active rheumatic infection.

Dr. Bland: The aortic valve was not involved?

Dr. Castleman: It showed a little calcification on the sinus side of the valve but no true interadherence. I think one would question whether there was any.

Dr. Bland: Was there any pericarditis?

Dr. Castleman: No.

Dr. Sylvester McGinn: How about the leg veins?

Dr. Castleman: The source of the infarct in the lung was the left popliteal vein. It is often supposed that the infarcts in the lungs in patients with fibrillation are due to mural thrombi in the right auricle, but if one examines the deep leg veins in those cases, another more likely source may be uncovered. In this case, the right auricle was normal.

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Erratum

In Case 27331 of the "Case Records of the Massachusetts General Hospital" in the August 14 issue of the *Journal* (line 18, column 1, page 272), the word "aminophyllin" was used erroneously for aminopyrine. The speaker did not mean to imply that aminopyrine caused liver damage, but that it was an equally dangerous drug, the toxicity in this case being directed toward the granulocytes.

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PUBLIC EDUCATION IN MEDICAL MATTERS

The Diabetes Education Program that is being sponsored by the Metropolitan Life Insurance Company is an encouraging example of public interest in good medicine. The increasing evidence that the early recognition and careful control of diabetes are important in checking the progress of the pancreatic and neurovascular lesions makes such a program particularly timely.

The economic value of good health to such an insurance company is of course obvious. But good public health or that of the individual is of equal value to private enterprise. Loss of work due to ill health is an economic burden to almost all industry. The vast sums of money spent annually by the American people on life and sickness insurance provide evidence concerning the value that they have long placed on life and health. Advances in medical science have so increased the effectiveness of available medicine that the public's interest is shifting from *insuring* health and life by cash indemnities to *securing* health and longevity by medical care. The expansion of health programs by state departments of public health and a variety of nonprofit lay organizations reflects the public's increasing appreciation of the effectiveness of modern medicine. The National Health Conference and the recent National Nutrition Conference were reflections of public awareness and interest. The federal government has responded through the programs of the United States Public Health Service, of the Maternity and Child Welfare Division of the Department of Labor, and of the National Health Institute at Bethesda, Maryland. This increased awareness is creating an increased demand for medical care. To finance this care, a variety of plans for voluntarily distributing and budgeting the costs of medical care are developing throughout the country. Although it is not widespread, there is even some demand for compulsory health insurance. Insofar as the layman's medical education is good and the plans for budgeted health services are sound, the medical profession, as well as the public, will benefit. The result should be more and better medicine more widely and, therefore, with present economic trends, better paid for in the long run.

So far so good, and the medical profession should indeed be happy. But there are, of course, many who will turn the public's interest in medicine to their personal advantage. For every educational program like that sponsored by the Metropolitan Life Insurance Company for better health, there will be one hundred programs on the radio and in the press sponsored by commercial organizations for the sale of unneeded or worthless medication. The same American advertising genius that has popularized alkalizing, laxatizing and antihalitozing remedies apparently is taking over the newly cultivated fields of vitamin and hormone

therapy. As it does so, the money that the public should spend for effective health and medical services will be squandered to such an extent that sums spent on the trial-and-error method of establishing workable insurance schemes will seem inconsequential. But no matter what becomes of the money, the medical profession in the end will pay the bill, so the public will be happy anyway.

MEDICAL PATENTS

THE medical profession has always been imbued with the idea of the free exchange of knowledge gained from research. The meetings of our societies and the contents of our journals bear witness to this fact. The inauguration of medical patents in certain fields has cast a blight on this traditional ideal. Holders of medical patents are learning that the onerous duties of controlling and protecting their patents tend to cast disturbing shadows over the glory of discovery.

The Department of Justice recently chose to bring suit against three leading pharmaceutic manufacturers in the United States on the charge of fixing the price of insulin in violation of the Sherman Act. Heavy fines were imposed. The University of Toronto, holders of the patent, had granted licenses to these manufacturers to make and sell insulin. The Insulin Committee of this institution has apparently carried out its responsibilities concerning standards and prices of the product marketed in a manner satisfactory to the medical profession of North America, and this committee has never ventured to control research in the field of diabetes. The judgment, then, seems to be against the manner of price fixing rather than against the quality of the product or any unreasonable profit on the part of the manufacturers.

The emoluments received by the patentees in this case were beyond the scope of this judgment. At one time, the royalties on insulin were 10 per cent, whereas those on the Dick patent, held by a committee associated with the University of Chicago, were 5 per cent. Yet, on June 4, 1935, the Council of the Massachusetts Medical Society adopted a resolution expressing disapproval of the Dick patent, the objection being based purely on the ground of the patentees' monopolistic control of investigative work in the field of scarlet fever.

Since patents appear to be necessary for the control of standardization of products in the existing situation, the control should lie with the highest tribunal of our medical councils or with the Government. The patents would then be dedicated to the public, and all royalties abolished in conformity with a tradition handed down from the days of Hippocrates. This suggestion was presented more than a year ago before the House of Delegates of the American Medical Association, but was set aside. It behooves the American Medical Association to renew its interest in the solution of this problem before federal authorities, probing farther into medical patents, charge organized medicine with lending support to a monopolistic practice that strikes at the very heart of academic freedom.

MEDICAL EPONYM

KOCH'S POSTULATES

These postulates are laid down in the monumental contribution of Robert Koch (1843–1910), "Die Aetiologie der Tuberculose (The Etiology of Tuberculosis)," which was delivered as a lecture to the Physiological Society in Berlin on March 24, 1882. It appears in the *Berliner klinische Wochenschrift* (**19:** 221–230, 1882). A portion of the translation follows:

> To prove that tuberculosis is a parasitic disease occasioned by the invasion of the bacilli and primarily caused by their growth and increase, these bacilli had to be isolated from the body, and kept in pure culture until they were free from any possible contamination with other disease products of the animal organism; and finally it was necessary to reproduce the same disease picture of tuberculosis (which experience showed could be obtained by injection of naturally produced tuberculous material) by transfer of the isolated bacilli into animals.

Koch then described his method of staining the tubercle bacilli, finding them in the lesions and culturing them. His success in reproducing tuberculous lesions and recovering the bacilli from them established a standard for this type of bacterial investigation that has remained fixed ever since.

R. W. B.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY*

Albuminuria and Hypertension Followed by Post-Partum Eclampsia and Death

A thirty-one-year-old para III was seen in the fifth month of pregnancy for the first time. Following that, she was seen by the district nurse quite frequently.

The past history was not remarkable. Physical examination at the time of her visit to the physician was reported to have been negative.

Six weeks before delivery, the patient had a systolic blood pressure of 150. During the next month, she was seen ten times by the district nurse, who found that the systolic blood pressure averaged from 150 to 200 and that there was a trace of albumin in the urine. The attending physician was notified of these findings. The patient started in spontaneous labor at term and delivered herself of a living child. An hour later, several severe convulsions occurred. The patient was sent to the hospital and died approximately one day after delivery, without regaining consciousness.

Comment. Albuminuria and increased blood pressure during pregnancy, and convulsions following delivery, still occur in this day of enlightened medicine. There is no reason to suppose that this death was unavoidable. Any conscientious practitioner, when notified that a patient had a systolic blood pressure of 200 and a trace of albumin a few weeks before the expected date of delivery, would have done something about the situation. Such a patient should be hospitalized and intensively treated by a milk diet and milk of magnesia; salt should be omitted from the diet. If the blood pressure does not come down, labor should be induced, thus preventing a fatality. This case is a disgrace to the medical profession.

*A series of selected case histories by members of the section will be published weekly. Comments and questions by subscribers are solicited and will be discussed by members of the section. Letters should be addressed to Dr. Raymond S. Titus, *Secretary*, 330 Dartmouth Street, Boston.

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DEATHS

POWERS — GEORGE H. POWERS, M.D., of Boston, died October 4. He was in his sixty-fifth year.

Born in San Francisco, Dr. Powers received his degree from the University of California Medical School in 1902. He was a fellow of the Massachusetts Medical Society and the American Medical Association, and was a member of the American Board of Otolaryngology and the American Otological Society.

He is survived by his widow and two daughters.

ROGERS — MARK H. ROGERS, M.D., of Boston, died October 5. He was in his sixty-fifth year.

Born in South Sudbury, Dr. Rogers graduated from Williams College in 1900 and received his degree from Harvard Medical School in 1904. He was professor of orthopedic surgery at Tufts College Medical School, chief

of the orthopedic service at the Beth Israel Hospital and orthopedic surgeon at the New England Deaconess Hospital. He was a fellow of the Massachusetts Medical Society, the American Medical Association and the American College of Surgeons.

His widow, a daughter, a son and two grandchildren survive him.

BOOK REVIEWS

Papers of Walter Hampton Frost, M.D.: A contribution to epidemiological method. Edited by Kenneth F. Maxcy, M.D. 8°, cloth, 628 pp. New York: The Commonwealth Fund, 1941. $3.00.

Frost, at the time of his death in 1938, was one of the leading epidemiologists in America. Brought up in the United States Public Health Service, a ground school for practically every important epidemiologist developed in this country, he was later chosen by William H. Welch to serve in the new School of Hygiene and Public Health established in 1919 at Johns Hopkins University. His previous work, particularly his accepted studies of acute anterior poliomyelitis in Iowa, Ohio and New York, which were considered among the outstanding reports on the disease in this country, and his other studies on typhoid fever, sewage pollution and influenza had given him a world-wide reputation. After becoming established at Johns Hopkins, he showed great interest in tuberculosis, partly, no doubt, because he suffered from this disease himself, and papers on this subject, in addition to the contributions on the principles of public-health practice, formed the bulk of his work during the latter years of his life. As these papers were widely scattered in medical periodicals, a committee of some of his friends and associates was formed in 1938, with Dr. K. F. Maxcy and others, to publish them in book form. The project had the sympathetic support of the Commonwealth Fund and a volume of over 600 pages is now available. The papers, in general, have been kept in their original form, although most of the charts have been redrawn for uniformity. Dr. Maxcy has written a brief but stimulating introduction giving the chief facts about the life of Frost and an estimation of his scientific contributions and character. The frontispiece is an excellent reproduction of a photograph of Frost, and the whole volume is superbly printed. Rarely does one meet in medical publications such a satisfactory and fitting format. The frame, indeed, complements the picture, and the picture itself is of one of America's great epidemiologists. The world of medical literature has been enriched by the devotion of Dr. Maxcy to his predecessor and the farsightedness of the Commonwealth Fund in seeing that this book was published.

Infantile Paralysis: Anterior poliomyelitis. By Philip Lewin, M.D. 8°, cloth, 372 pp., with 165 illustrations. Philadelphia: W. B. Saunders Company, 1941. $6.00.

This book is a careful review of all aspects of poliomyelitis, with particular emphasis on orthopedic aftercare. Written by an orthopedic surgeon of note, this important aspect of the treatment of poliomyelitis receives adequate attention, with numerous illustrations of operative procedures. In the earlier chapters, however, on etiology, portals of entry, resistance and immunity, epidemiology and similar topics, as well as in those on examination and diagnosis, these points do not appear to be adequately covered. The ideas are sound, and the statements accurate, in general, although some may disagree with the opinion expressed in the chapter on diagnosis. The book can nevertheless be highly recommended as an excellent brief account of a disease that is ever present and is a constant menace. This is a practical book of particular value to orthopedic surgeons and to the general practitioners who wish to understand the mechanics of orthopedic appliances and surgical procedures.

Modern Drugs in General Practice. By Ethel Browning, M.D., Ch.B. 8°, cloth, 236 pp. Baltimore: Williams and Wilkins Company, 1940. $3.00.

This is a list of drugs in common use, with a brief bibliography attached to each chapter. In most cases, the name of the drug, as supplied in Great Britain, is added, since the book emanates from British sources. It is a valuable, up-to-date listing of current pharmaceuticals, briefly presented, giving details about the drugs themselves, their clinical indications, dosage, toxic effects, the mode of action and the results of treatment. The book should prove to be of some value to American physicians, but other books of equal value, written from the American standpoint, are available.

NOTICES

ANNOUNCEMENT

Dr. Franz R. Stenzel announces the removal of his office from 1580 Beacon Street, to 1801 Beacon Street, Waban.

SOUTH END MEDICAL CLUB

A meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, October 21, at 12 m. Dr. Donald E. Currier, chief of the Medical Division of the Massachusetts Selective Service System, will speak on "The Physician in National Defense."

Physicians are cordially invited to attend.

JOHN T. BOTTOMLEY SOCIETY

The fourth annual clinical meeting of the John T. Bottomley Society will be held at the Carney Hospital on Wednesday, October 22, at 10:00 a.m. Luncheon will be served at 1:00 p.m. The annual dinner will be held at the Copley-Plaza at 6:30 p.m.

Program

Erythroblastosis Fetalis. Dr. William C. Moloney.

Recent Advances in the Treatment of the Menopause. Dr. John J. Thornton.

The Management of the Acute Gall Bladder. Dr. William J. Sullivan.

Common External Diseases of the Eye. Dr. George R. Gagliardi.

Recent Advances in the Treatment of Skin Diseases. Dr. John T. Foley.

NEW ENGLAND PATHOLOGICAL SOCIETY

A meeting of the New England Pathological Society will be held at the Boston Lying-in Hospital on Thursday, October 16, at 8 p.m.

Program

Morphologic Findings in Endometrial Biopsies from 100 Cases of Bleeding during the First Trimester of Pregnancy and from 34 Postabortal and Normal Puerperal Uteri. Dr. Robert N. Rutherford.

Bilateral Cortical Necrosis of the Kidney: A report of 2 cases, with some observations on the pathogenesis and evolution of the renal lesion. Dr. Walter H. Sheldon.

The Relation of Ovarian Stromal Hyperplasia and Thecoma of the Ovary to Endometrial Carcinoma. Dr. Lent C. Johnson.

Observations on a Series of Early Normal and Abnormal Human Ova Obtained Prior to the First Missed Menstrual Period. Dr. A. T. Hertig.

Business meeting.

Collation.

NEW ENGLAND SOCIETY OF PSYCHIATRY

The regular fall meeting of the New England Society of Psychiatry will be held at the Augusta State Hospital, Augusta, Maine, on Friday, October 17. Dr. Donald E. Currier, chief of the Medical Division, Massachusetts Selective Service System, will speak on "Medical and Psychiatric Features of the Selective Service Program."

AMERICAN CONGRESS ON OBSTETRICS AND GYNECOLOGY

The second American Congress on Obstetrics and Gynecology will be held in St. Louis, Missouri, April 6 to 10, 1942. All the meetings and the commercial, educational and scientific exhibits will be held in the Public Auditorium.

The general plan for the program will be much the same as that of the first congress, which was held in Cleveland in 1939, with sectional meetings for the various groups (nurses, public-health officers, administrators, educators and physicians), general sessions for all members attending the congress and round-table discussions. The evening sessions will be open to the general public.

Admission to the congress will be by membership cards only, which may now be secured by the payment of a $5.00 registration fee. All mail should be addressed to and further information may be obtained from the American Congress on Obstetrics and Gynecology, 650 Rush Street, Chicago.

UNITED STATES CIVIL SERVICE EXAMINATION

Maternal and Child-Health Specialists

Employment registers are to be established by the Civil Service Commission to fill maternal and child-health specialist positions in the Children's Bureau of the Department of Labor. Vacancies in similar positions in state agencies co-operating with the Children's Bureau may also be filled from these registers at the request of the states concerned. The examination announcement just issued by the Civil Service Commission to recruit persons for these positions allows the filing of applications until November 15.

There are three options in which persons may qualify: pediatrics, obstetrics and orthopedics. For each of these, employment lists will be established for administrative, research and clinical positions. The duties of the administrative positions include giving consultations and advisory service to state and other governmental agencies carrying out maternal and child-health programs. The research positions involve the planning or directing of studies in such fields as infant and maternal mortality, and child growth in relation to social, economic and other factors. Persons appointed to clinical positions will do clinical work in one of the options.

A written test will not be given for these positions. Competitors will be rated on their education, experience and corroborative evidence. Applicants must have graduated from a medical school of recognized standing and must have served a one-year internship. In addition, they must have had full-time postinternship clinical training, as well as other appropriate experience in the option selected and in the type of work in which they seek appointment.

Physicians who are interested in this opportunity are urged to seek further information about these positions, which pay from $3200 to $5600 a year. Further information and application forms may be obtained from the commission's representative at any first-class or second-class post office or from the central office in Washington, D. C.

SOCIETY MEETINGS AND CONFERENCES

Calendar of Boston District for the Week Beginning Sunday, October 12

Monday, October 13

12:15–1:15 p.m. Clinicopathological conference. Peter Bent Brigham Hospital amphitheater.

Tuesday, October 14

*9–10 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

12:15–1:15 p.m. Clinicoroentgenological conference. Peter Bent Brigham Hospital amphitheater.

8:15 p.m. Harvard Medical Society. Peter Bent Brigham Hospital amphitheater.

Wednesday, October 15

*9–10 a.m. A Dental Pediatric Presentation. Dr. F. C. McDonald and Dr. Leonard Despres. Joseph H. Pratt Diagnostic Hospital.

*12 m. Clinicopathological conference. Children's Hospital.

Thursday, October 16

*8:30 a.m. Combined clinic of the medical, surgical, orthopedic and pediatric services of the Children's Hospital and the Peter Bent Brigham Hospital, at the Children's Hospital.

*9–10 a.m. Medical clinic. Dr. S. J. Thannhauser. Joseph H. Pratt Diagnostic Hospital.

8 p.m. New England Pathological Society. Boston Lying-in Hospital.

Friday, October 17

*9–10 a.m. Gastric Ulcer. Dr. Sara Jordan. Joseph H. Pratt Diagnostic Hospital.

Saturday, October 18

*9–10 a.m. Presentation, with discussion, dispensary and district cases. Joseph H. Pratt Diagnostic Hospital.

*Open to the medical profession.

October 9–May 14. Pentucket Association of Physicians. Page 473, issue of September 18.

October 11. American Social Hygiene Association. Page 559, issue of October 2.

October 13–24. 1941 Graduate Fortnight of the New York Academy of Medicine. Page 834, issue of May 8.

October 14–17. American Public Health Association. Page 579, issue of March 27.

October 17. New England Society of Psychiatry. Notice above.

October 19–23. American Academy of Ophthalmology and Otolaryngology. Page 350, issue of August 28.

October 21. South End Medical Club. Page 599.

October 22. John T. Bottomley Society. Page 599.

October 29–30. New England Postgraduate Assembly. Pages ii-iii, issue of September 11.

October 29–November 1. Association of Military Surgeons. Page 473, issue of September 18.

October 30. Journal Club meeting, Boston Lying-in Hospital, 8 p.m.

November 3–7. American College of Surgeons. Page vii, issue of July 31.

November 5–6. American Conference on Industrial Health. Page 473, issue of September 18.

January 3. American Board of Obstetrics and Gynecology. Page 473, issue of September 18.

January 10–11. Forum on Allergy. Page 392, issue of September 4.

April 6–10. American Congress on Obstetrics and Gynecology. Notice above.

April 20–24. American College of Physicians. Page 996, issue of June 5.